ARCHIVES OF

NEUROLOGY AND PSYCHIATRY

VOLUME 16
1926

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

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VOLUME 15 NUMBER 1

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

JULY, 1926

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, Jan. 7, 1919, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized Jan. 15, 1919.

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JUNE, 1926. NUMBER 6

Archives of Neurology and Psychiatry

VOLUME 16 JULY, 1926 NUMBER 1

STUDIES ON THE CENTRAL VISUAL SYSTEM

I. THE ANATOMIC PROJECTION OF THE RETINAL QUADRANTS ON THE STRIATE CORTEX OF THE RABBIT *

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BOSTON

An understanding of the relations of the central visual system of animals will doubtless aid our appreciation of the precise conditions in the human brain. The visual pathways of animals can be accurately determined by the study of experimentally produced degenerations, as is shown by the experiments of Minkowski [1] on the geniculocortical connections in dogs and cats and the experiments of Brouwer [2] and Zeeman [3] on the representation of the retinal quadrants in the corpus geniculatum laterale and anterior corpus quadrigeminum of the rabbit, cat and ape. In order to complete our knowledge of the anatomy of the visual system in the rabbit, the experimental investigation of the projection of the corpus geniculatum on the cortex seemed desirable.

It is unnecessary to review completely the history of the discovery of the central visual connections in animals, as this is well covered in the papers just cited. Apparently the first investigator to study the degenerations produced in the external geniculate body of rabbits after

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1. Minkowski, M.: Zur physiologie der Sehsphäre, Arch. f. Physiol. **141**:171, 1912; Experimentelle Untersuchungen über die Beziehungen der Grosshirnrinde und der Netzhaut zu den primären optischen Zentren, besonders zum Corpus geniculatum externum, Habilitationsschr. Zürich, 1913, p. 104.

2. Brouwer, B.: Experimentelle-anatomische Untersuchungen über die Projektion der Retnia auf die primären Opticuszentren, Schweiz. Arch. f. Neurol. u. Psychiat. **13**:118, 1923.

3. Brouwer, B., and Zeeman, W. P. C.: Experimental Anatomical Investigations Concerning the Projection of the Retina on the Primary Optic Centers in Apes, J. Neurol. & Psychopath. **6**:1 (May) 1925.

localized cortical lesions was von Monakow.[4] His experiments, which have become classic, served only to outline the general position of the visual cortex, however, and did not yield any precise information as to more detailed localization within this area. In the cat's brain, he found [5] the mesial portion of the cortical *"Sehsphäre"* represented laterally in the corpus geniculatum externum, the lateral portion mediofrontally, the posterior part laterally and posteriorly. But he carried correlation no further than this.

Van Valkenburg,[6] while engaged in the study of the origin of callosal fibers, noticed that small lesions of the striate region were followed by sharply localized degenerations in the external geniculate body of the cat. He, however, did not investigate the exact pattern of the projection further.

The experiments of Minkowski [1] show what detailed and precise information can be obtained by the study of experimental degenerations. In a series of five cats, areas as small as 0.5 cm. in diameter were excised from the striate cortex. In each case, sharply defined and consistent degenerations were produced in the corpus geniculatum. Schematized, his results showed that the anterior (and superior) portion of the visual cortex is represented in the anterior (and inferior) portion of the corpus geniculatum; the posterior (and inferior) portion of the cortex in the posterior (and superior) portion. The relations laterally and medially were not clearly defined. The enucleation of one eye showed that the temporal and nasal retinal halves are represented in different portions of the corpus geniculatum, but yielded no precise information as to the projection of the retina on the cortex. His experiments on dogs, in which the visual fields were tested by special methods, led to the conclusion that the superior retinal quadrant is represented in the anterior portion of the area striata, the inferior quadrant in the posterior portion; but unfortunately the brains of these animals were not studied microscopically.[1]

The first exact determinations of the projection of the retina on the primary visual centers are those of Brouwer and Zeeman,[2] which form

4. Von Monakow, C.: Ueber einige durch Exstirpation circumscripter Hirnrindenregionen bedingte Entwickelungshemmungen des Kaninchengehirns, Arch. f. Psychiat. u. Nervenh. **12**:141, 1881.

5. Von Monakow, C.: Experimentelle und pathologisch-anatomische Untersuchungen über die Beziehungen der sogenannte Sehsphäre zu den infracorticalen Opticuscentren und zum Nervus opticus, Arch. f. Psychiat. u. Nervenh. **14**:699, 1883; ibid. **16**:151 and 319, 1885.

6. Van Valkenburg, C. T.: De oorsprong der vezels in het corpus callosum en het psalterium, Verslag Koninklijke Academie van Wetenschappen te Amsterdam, 1911, p. 1.

the starting point for the present paper. Localized areas of the retina were incised under ophthalmoscopic observation, and the degeneration was followed through the nerve and tract to the corpus geniculatum externum and anterior corpus quadrigeminum of the opposite side.

The results in twelve experiments in rabbits were perfectly definite and consistent, and show the temporal half of the retina to be represented mainly on the mesial aspect of the ganglion, the nasal half on the lateral aspect. The upper half of the retina is represented mainly in the lower half of the corpus geniculatum, the lower half mainly above.

A similar exact projection was found to exist in the corpus quadrigeminum anterior, which need not concern us here. Four experiments were performed in cats, in which relations appear to be similar. More recently, Brouwer and Zeeman [3] have published the results of similar investigations in apes. Minkowski [7] states that he has performed localized extirpations in the striate cortex of this animal, but has not yet published a complete account of them.

Overbosch [8] has determined the extent and position of the projection of the binocular field of vision on the corpus geniculatum externum of rabbits and cats, by enucleating one eye and following the degenerations by means of the Marchi method. In his rabbit preparations, the degeneration involves only a small proportion of the fibers of the homolateral tract, but spreads out over perhaps an eighth or a tenth of the corresponding corpus geniculatum.

METHODS OF STUDY

The methods of study were the same in all cases. The operations were performed by Professor Brouwer at the Wilhelmina Gasthuis, Amsterdam. Healthy rabbits (*Lepus cuninculus*) were used. Under aseptic precautions a small trephine opening was made in the skull of the etherized animal, and a determined area of cortex was excised from the occipital region. All of the animals made an uneventful recovery, and all were killed at the end of ten months.

The brains were fixed in formaldehyde, and embedded in celloidin. Complete serial sections were made through the entire striate area, including the corpus geniculatum externum. Alternate sections were stained by the Weigert-Pal method and with van Gieson's stain.

In these sections, both the cortical lesion and the resulting atrophy in the corpus geniculatum could usually be outlined with certainty. In some

7. Minkowski, M.: Étude sur les connexions anatomiques des circonvolutions rolandiques, pariétales, et frontales, Schweiz. Arch. f. Neurol. u. Psychiat. **15**:7, 1924.

8. Overbosch, H. J.: Inaugural Diss., Amsterdam, to be published.

cases, however, the cortical lesion was so superficial that the cortical architecture could easily be recognized. The doubtful areas are represented in the diagrams of the cortex by coarse stippling. From a consideration of all the results taken together it seems probable that such superficial lesions usually do not cause atrophy of the corpus geniculatum. The deeper lesions often penetrated the ventricle and usually involved the radiation as well as the cortex. This must be considered in interpreting the results, but does not complicate them greatly. There was always some atrophy of the radiation, which could be identified by the coarseness of its fibers. But in only one instance (experiment 6) could the atrophic portion be followed as a distinct bundle.

The secondary changes in the corpus geniculatum externum consisted of a local proliferation of neuroglia, and a paling and decrease of the ganglion cells (figs. 4, 7 and 10). The extent of the degeneration was checked by several observers, and in case of doubt, the questionable area was counted as normal. Usually there were similar localized changes elsewhere in the thalamus, which need not concern us as they are outside the visual system. No changes were found in the anterior corpora quadrigemina, in the optic tracts or in the contralateral ganglion.[9] The ventral nucleus of the external geniculate body never showed definite atrophy.

In order to obtain an exact reproduction of the cortical lesion, its extent and the extent of the area striata were determined on sections taken at definite intervals throughout the series. These were then measured off with calipers on a diagram. As the limit between the areas 17 and 18 (Brodmann) is extremely difficult to determine accurately in the rabbit, both are included in the lined area in the diagrams. At most, area 18 is very small in the rabbit.

In order to obtain a three-dimensional conception of the extent of degeneration in the corpus geniculatum, the following method was devised. Every eighth section was taken from the series through the ganglion, and a projection sketch was made from it with a magnification of thirty times. The extent of degeneration was outlined on it, under microscopic control. By means of a drawing prism, successive sketches were drawn as if observed from an acute angle and superposed on one another. Thus, the outline of the degeneration on the surface of the ganglion is obtained, and also a suggestion of the depth to which the degeneration penetrates. The proportional dimensions of the geniculate

9. Von Monakow found atrophy of these structures after extirpations of the visual cortex. He also found lesions in the thalamus itself. His experiments were performed on new-born animals, however, and the cortical lesions were proportionally much larger than in our case.

body are approximately, but of course not absolutely, accurate in these diagrams.

This general description applies to all the experiments. The individual protocols may be given briefly.

PROTOCOLS OF EXPERIMENTS

1. *Lesions of the Anterior Portion of the Striate Area.*

Experiment 1.—A rabbit, aged 5 weeks, was operated on, July 14, 1922, and the anterior end of the striate cortex excised. At necropsy, April 11, 1923, the lesion was found to be superficial, and to extend somewhat forward of the striate region. The precise extent of the lesion, and the extent of the resulting degeneration are shown in figure 1, *A* and *B*. Both were sharply outlined.

Experiment 2.—A small rabbit, aged 2 months, was operated on, July 7, 1922. At necropsy, April 11, 1923, the lesion was localized with difficulty. It lay anteriorly, and was small, but entered the ventricle. The results of the microscopic examination are shown in figure 2, *A* and *B*. The cortical lesion does not extend to the mesial border of the striate area, but cuts the fibers of the radiation supplying it. The striate area has a slightly anomalous shape.

The atrophy in both these cases lies in the region of the corpus geniculatum in which binocular vision is represented (figs. 11 *A* and 12).

2. *Lesions of the Superior Portion of the Area Striata.*

Experiment 3.—A rabbit, aged 5 weeks, was operated on, July 14, 1922. Necropsy, April 11, 1923, showed the lesion to be large, including all of the superior border. It was ill-defined inferiorly. Sections showed the extent of damage to be limited to about the upper third of the striate area. In the ganglion, the degeneration included the area of representation of the binocular field and extended inferiorly and posteriorly (figs. 3, *A* and *B*, and 4).

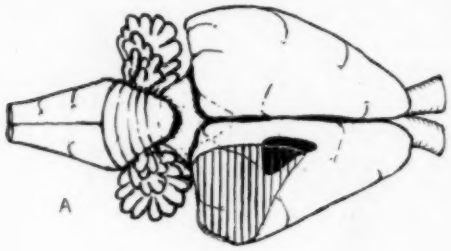
Experiment 4.—A rabbit, aged 5 weeks, was operated on, July 12, 1922. Necropsy showed an extensive, superficial lesion of the superior half of the striate area. Microscopic sections showed that the anterior and inferior portion of the lesion was so superficial as not to disturb the stripe of Gennari. This portion is stippled in figure 5, *A* and *B*. The lesion produced was little larger than that of the previous experiment, but extended above the binocular region also (fig. 5, *C*).

3. *Lesion of the Middle Portion of the Striate Area.*

Experiment 5.—Operation was performed July 14, 1922. The animal was killed, April 11, 1923. The lesion consisted of a deep cut, vertically across the area striata. Microscopic examination showed that the lesion spared the region which was excised in experiment 1. In the corpus geniculatum, there was also a small "island" of cells which still took a normal stain, although there was some proliferation of neuroglia about them (fig. 6, *B*, and 7). The position of this "island" corresponded roughly to that of the representation of the binocular field, and the degeneration fell chiefly in the area in which the temporal quadrants are represented (fig. 11, *A*). The radiation supplying other portions of the striate cortex did not appear to be disturbed.

4. *Lesions of the Posterior and Inferior Portions of the Striate Area.*

Experiment 6.—Operation was performed July 14, 1922. Necropsy, April 11, 1923, showed a large, ill-defined superficial lesion. In sections, the precise extent of the cortical lesion was determined with difficulty. The regions in which the



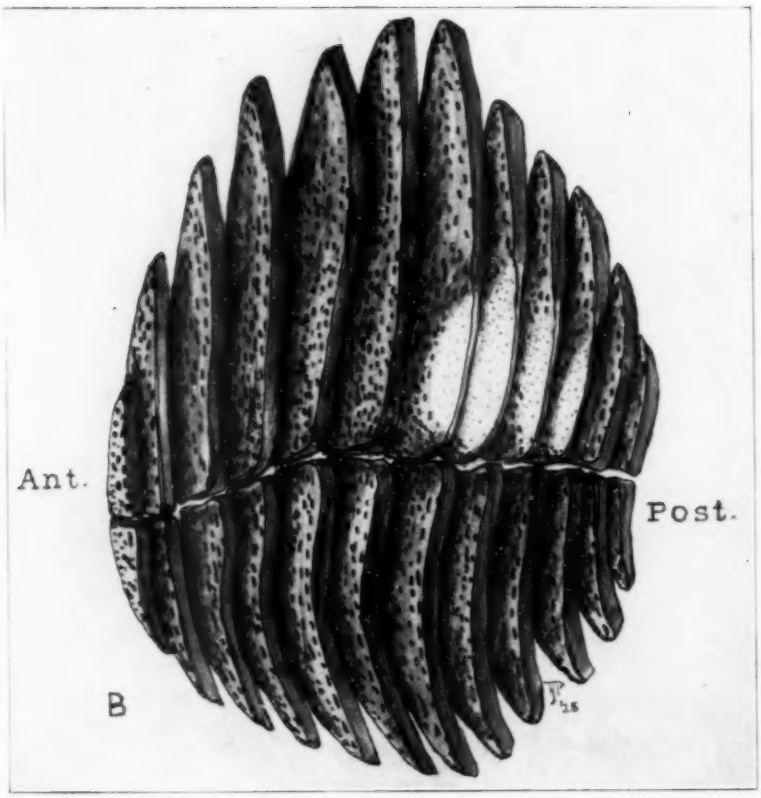
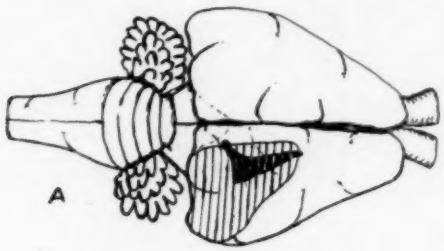


Fig. 1 (Experiment 1).—*A* is the diagram of a small lesion (in black) of the anterior end of the right striate cortex (outlined and striped) of the rabbit's brain, slightly enlarged; *B*, virtual reconstruction of the right external geniculate body, seen from the mesial side; successive sections are drawn with the camera lucida as if seen obliquely from the inner edge; normal tissue is shaded with coarse stippling; the degenerated area is shown in white, with fine stippling, lying about the middle of the mesial surface of the posterior half of the ganglion and it is superficial; the ventral nucleus shows no degeneration; semischematic; × 30.



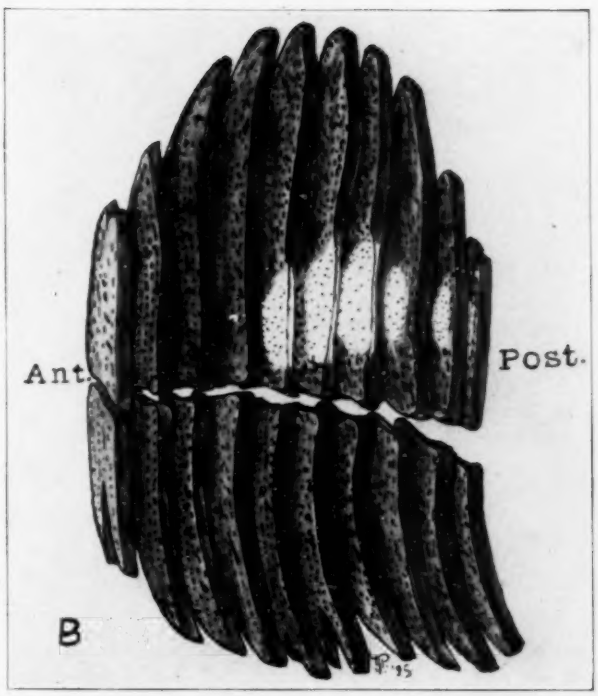
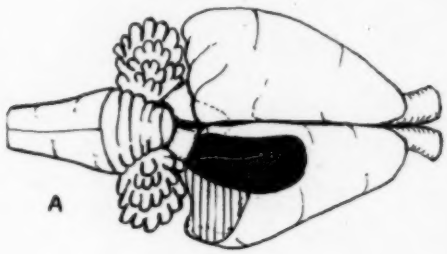


Fig. 2 (Experiment 2).—*A* is the diagram of a somewhat larger lesion (black) of the right striate cortex (striped); this was a small animal, and the striate area occupied a larger proportion of the brain than usual; *B*, virtual reconstruction of the right corpus geniculatum externum seen from the mesial side; the lesion (white, finely stippled) occupies a somewhat smaller area than the one in the previous case, but in the same region; this corresponds approximately to the situation of the representation of binocular vision (fig. 11, *A*, and 12); the ventral nucleus is undisturbed; × 30.



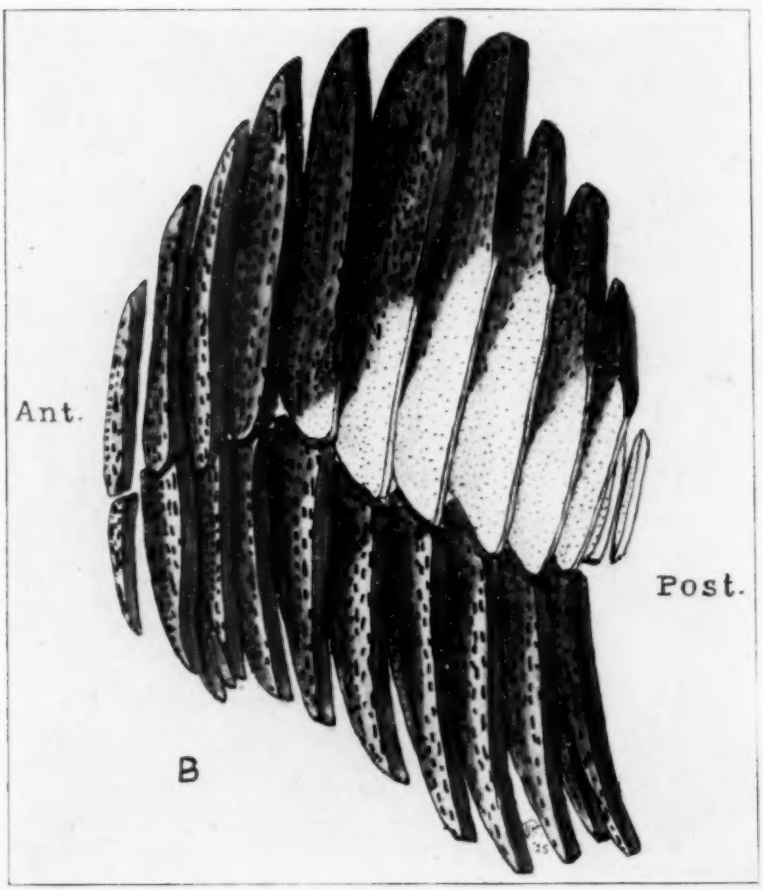


Fig. 3 (Experiment 3).—*A* is a large lesion of the superior part of the right striate cortex, slightly enlarged; *B*, virtual reconstruction of the right corpus geniculatum externum; the lesion involves the lower half of the mesial and almost all of the inferior surface of the posterior half of the ganglion; it includes the larger part of the areas of representation of both superior retinal quadrants, and of the binocular field (figs. 11, *A*, and 12); the ventral nucleus is undisturbed; × 30.

Fig. 4 (Experiment 3).—Photomicrograph of a section through the center of the degenerated area in the right corpus geniculatum; extent of degeneration indicated by arrows; atrophy of cells and proliferation of neuroglia should be noted; there is also a neuroglial proliferation in the optic radiation as it passes through the lateral thalamic nucleus (in the upper left hand corner); the ventral nucleus (below) is undisturbed; Van Gieson stain; × 45.

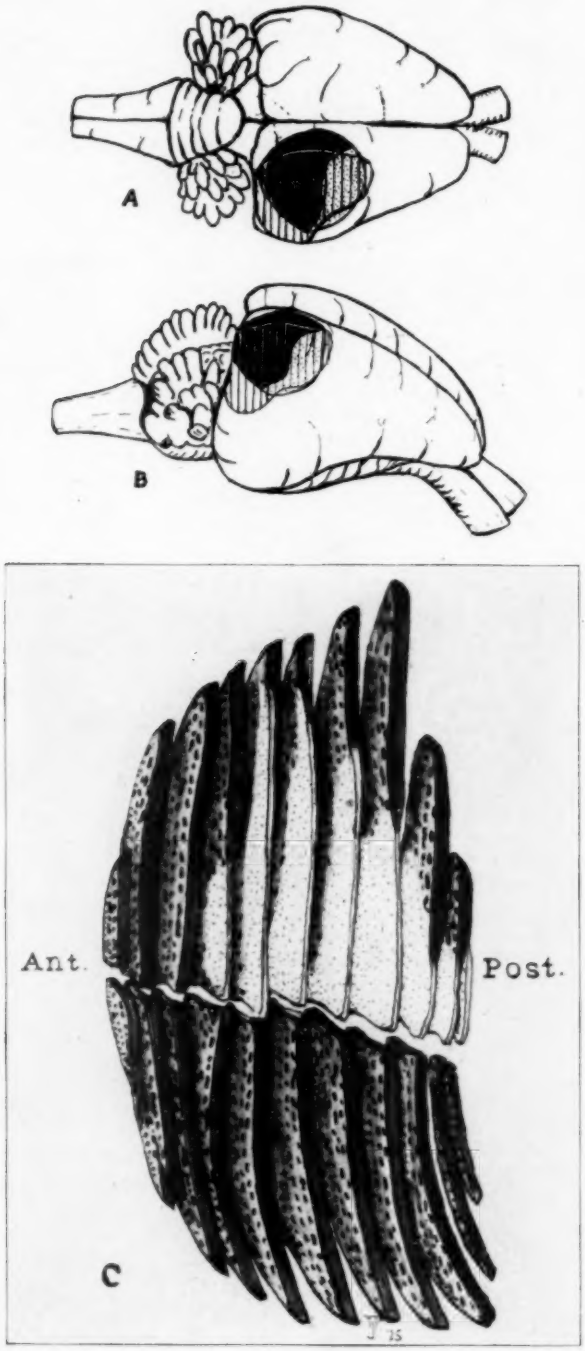


Fig. 5 (Experiment 4).—*A* is an extensive lesion (black) of the upper part of the striate cortex (striped); the anterior portion of the lesion (stippled) is superficial, and should probably be disregarded; the brain is viewed from above; slightly enlarged; *B*, diagram of the same brain, viewed more from the right side; slightly enlarged; *C*, virtual reconstruction of the right corpus geniculatum, seen from the mesial side; extensive degeneration of the mesial and inferior portions of the dorsal nucleus, extending deep into its substance; × 30.

degeneration was not definite are stippled in figure 8, *A*. Certainly, however, there was a degeneration of the posterior and inferior portion of the optic radiation. The fibers of the radiation supplying the interval between the two deep lesions must have been severed. The degeneration in the corpus geniculatum externum is shown in figure 8, *B*.

Experiment 7.—A rabbit, aged 5 weeks, was operated on, July 12, 1922. Necropsy, April 11, 1923, showed a deep lesion, limited to the posterior half of the striate area. The lesion had very definite outlines, which are shown in figure 9, *A*. A localized degeneration of the optic radiation was not observed. In the corpus geniculatum the degeneration lay chiefly on the lateral surface (fig. 9, *B*, and 10), corresponding in general to the position of the two nasal quadrants.

COMMENT

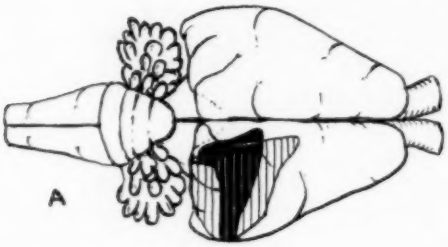
The shape of the area striata is appreciated with difficulty in gyrencephalic animals. It is plainly seen only in the lissencephalic. In the rabbit, it has the shape of a circle or oval, with a distinct outgrowth frontally. In still lower animals (*Erinaceus europaeus,* for instance) in which the visual system is very primitive, this irregularity is absent, as may be seen from the charts of Brodmann and others. It is tempting to suppose, *a priori,* that the specialized function of binocular vision is located in the newly acquired field.

If we may continue our deductive reasoning further, we may expect to find the region of binocular vision lying at the edge of the field representing the temporal half of the retina, as it is formed by overlapping of the two temporal fields. The representation of the nasal quadrants should lie on the other side of the temporal representation.

Perhaps the area allotted to the nasal quadrants may be smaller than that occupied by the temporal quadrants, and perhaps the superior quadrants will occupy less space than the inferior quadrants, as the animal looks mainly forward and upward.

With these suggestions in mind, we may proceed to the attempt to fit together the results of the experiments just described. But a consideration of the diagrams shows that the lesions are too few in number, and overlap too much, to give the result directly. We may, however, divide them further into smaller fields, by making a schema of the different areas in which two or more of them overlap. Such an attempt has been made in figures 11, *A* and *B,* and 12, in which the subdivided areas have been superposed on a diagram of the representation of the retinal quadrants in the corpus geniculatum, based on the work of Brouwer and Zeeman,[2] and of Overbosch.[8]

Plotting the corresponding areas on a diagram of the striate area, we have figure 13. It is not difficult to recognize the position of the temporal and nasal quadrants, and of the field of binocular vision. The



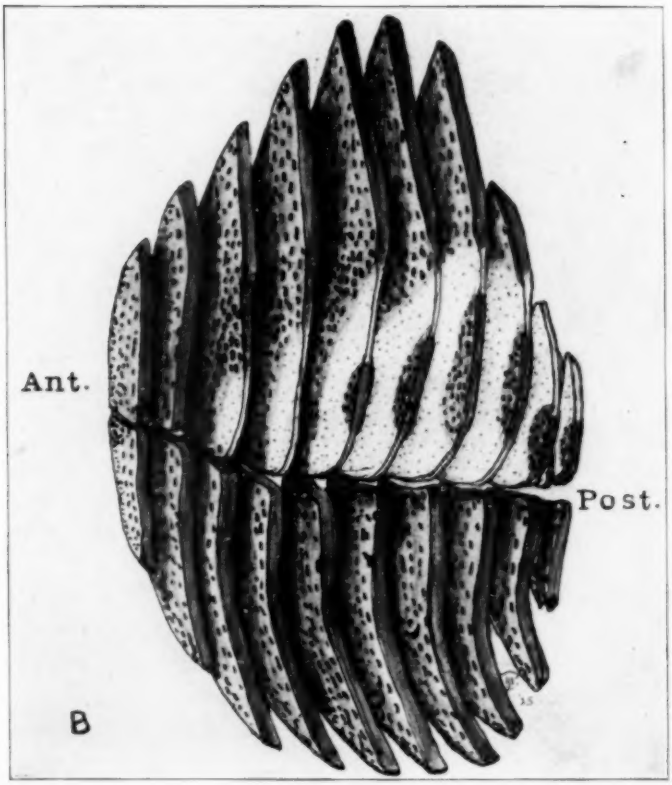


Fig. 6 (Experiment 5).—*A* is the diagram of a vertical lesion bisecting the striate cortex; this lesion extended deeply, cutting the optic radiation; slightly enlarged; *B*, virtual reconstruction of the right corpus geniculatum externum, viewed from the mesial side; the atrophy extends more deeply posteriorly than in the last case and reaches the lateral surface in some places; a small "island" of tissue is spared, in the region of the representation of the binocular field of vision; × 30.

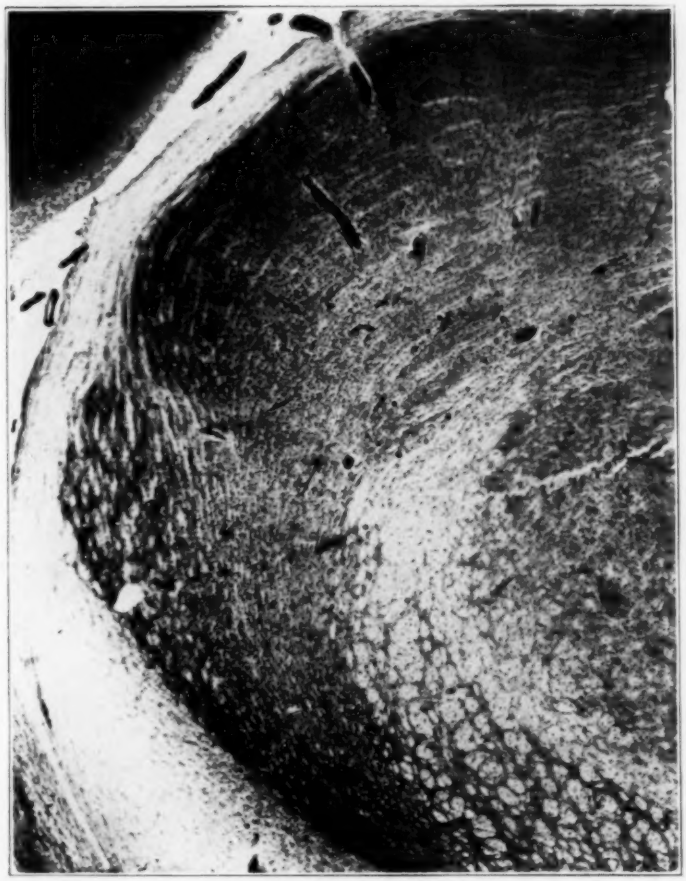
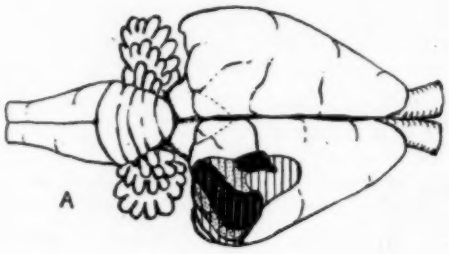


Fig. 7 (Experiment 5).—Photomicrograph of a section through the degenerated area (indicated by arrows); in the region outlined by black dots, there is a proliferation of neuroglia, but the cells are intact; there is also gliosis of the optic radiation and of the lateral thalamic nucleus; the ventral nucleus is intact; Van Gieson stain; × 45.



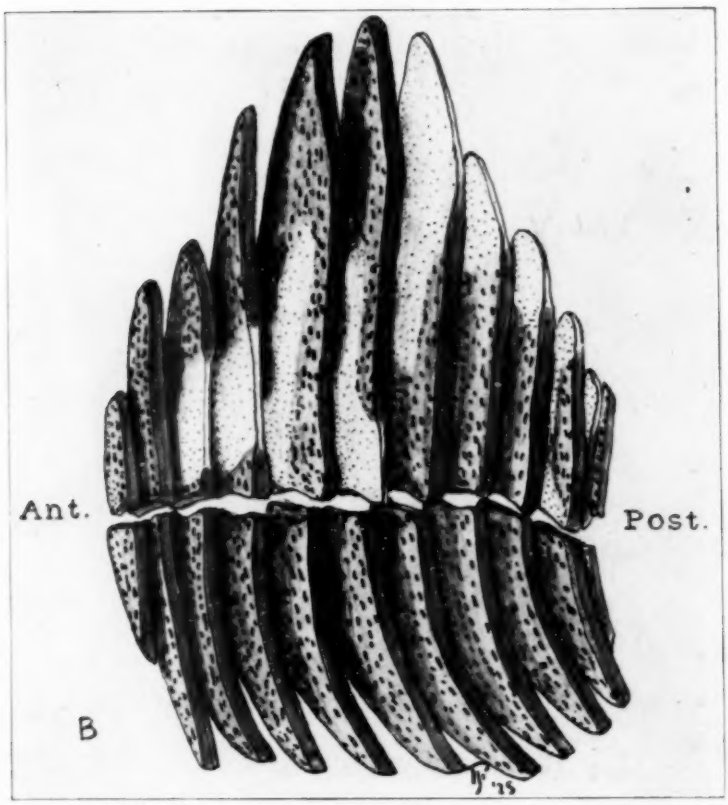


Fig. 8 (Experiment 6).—*A* is the diagram of an extensive lesion of the area striata; the inferior portion and the middle portion of the lesion (stippled) are superficial, and probably do not destroy the geniculocortical neuron; but the fibers going to the region between the two deep (black) portions of the lesion are doubtless severed; in addition an atrophy of the posterior fibers of the optic radiation was visible in sections; *B*, virtual reconstruction of the right corpus geniculatum externum, viewed from the mesial side; the atrophy is seen to lie in the inferior portion of the anterior half and the superior portion of the posterior half; the outlines of the lesion were less definite than usual; × 30.

exact boundaries are difficult to determine, but their mutual relationship seems definite, and falls in with our presupposed scheme. There can be little doubt that in rabbits the projection of the retina on the cortex, as well as on the corpus geniculatum, is anatomic and invariable.

In addition to the position of the representation of the binocular field its size is of interest. The proportion of the optic nerve which it occupies is small—not more than a twenty-fifth of the whole. In the corpus geniculatum, it takes up perhaps an eighth or less of the volume of the ganglion. But in the cortex, it occupies a fifth or more of the entire striate area.

It is of interest that the central optic pathways undergo an almost complete rotation in the rabbit, so that the superior retinal quadrants, which are represented in the inferior portion of the ganglion, are projected on the superior portion of the cortex. In addition, the field of binocular vision is represented mesially and posteriorly in the geniculate body, anteriorly on the cortex. It is not easy to carry the comparison between the three-dimensional ganglion and the two-dimensional cortical surface much further.

The binocular field is represented in the anterior portion of the optic radiation.

Other aspects of the problem of the central visual connections will be presented in a later paper. It is an extremely agreeable duty to thank Professor Brouwer for assigning us this problem in a field which has been so much enriched by his own investigations. He supplied not only the materials and opportunities for study, but also constant aid and encouragement. What there is of worth in the result is to be ascribed to him.

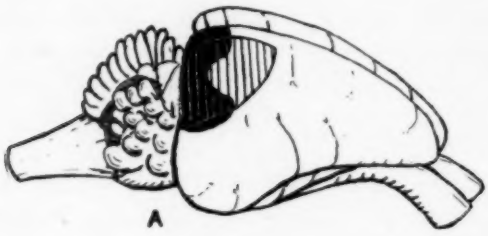
CONCLUSIONS

1. In the rabbit, there is a fixed and definite anatomic projection of the dorsal nucleus of the corpus geniculatum externum on the cortex. Lesions of the striate cortex only a few millimeters in diameter cause circumscribed and sharply localized areas of atrophy in the external geniculate body.

2. Contiguous lesions of the cortex produce contiguous lesions in the corpus geniculatum, and the size of the one is proportional to the size of the other, in the areas devoted to monocular vision.

3. Lesions of the striate cortex in adult rabbits cause no demonstrable lesions of the ventral nucleus, the anterior colliculus or the optic tract.

4. When the results of these experiments are combined with the already known projection of the retina on the corpus geniculatum, the nasal quadrants are found to be projected on the postero-inferior portion



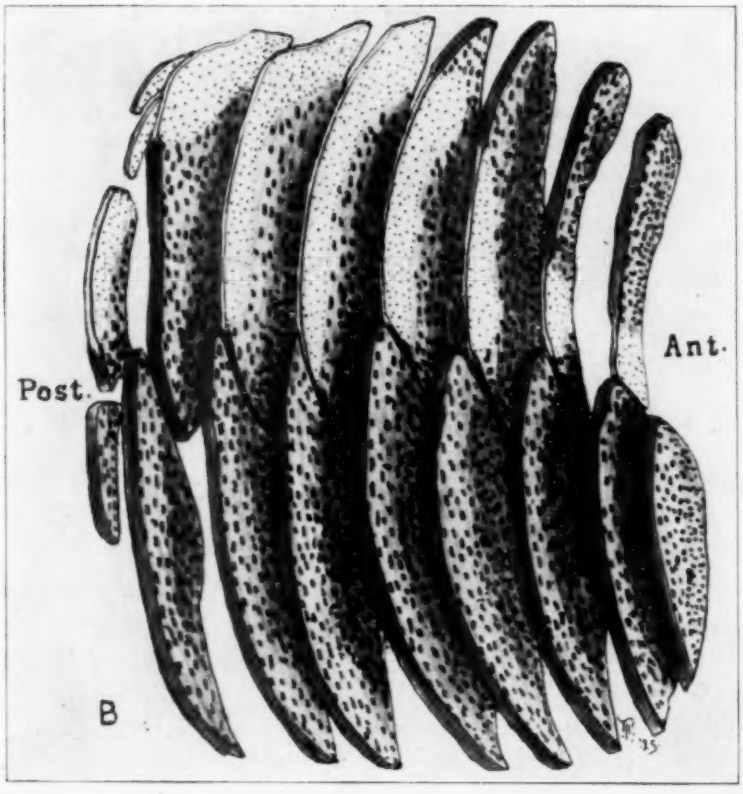


Fig. 9 (Experiment 7).—*A* is the diagram of a deep lesion involving the posterior portion of the right striate cortex; the anterior limit of the striate area was difficult to determine; the brain is viewed from the side; slightly enlarged; *B*, virtual reconstruction of the right external geniculate body, viewed from the lateral surface (unlike the other diagrams in the series); the degeneration lies exclusively on the lateral and superior surface of the ganglion, reaching the mesial surface only at the anterior end; × 30.

Fig. 10 (Experiment 7).—Photomicrograph of a section through the center of the ganglion. The degeneration (indicated by arrows) lies along the lateral and superior surface; the ventral nucleus is spared; there is no gliosis of the lateral thalamus nuclei or of the optic tract.

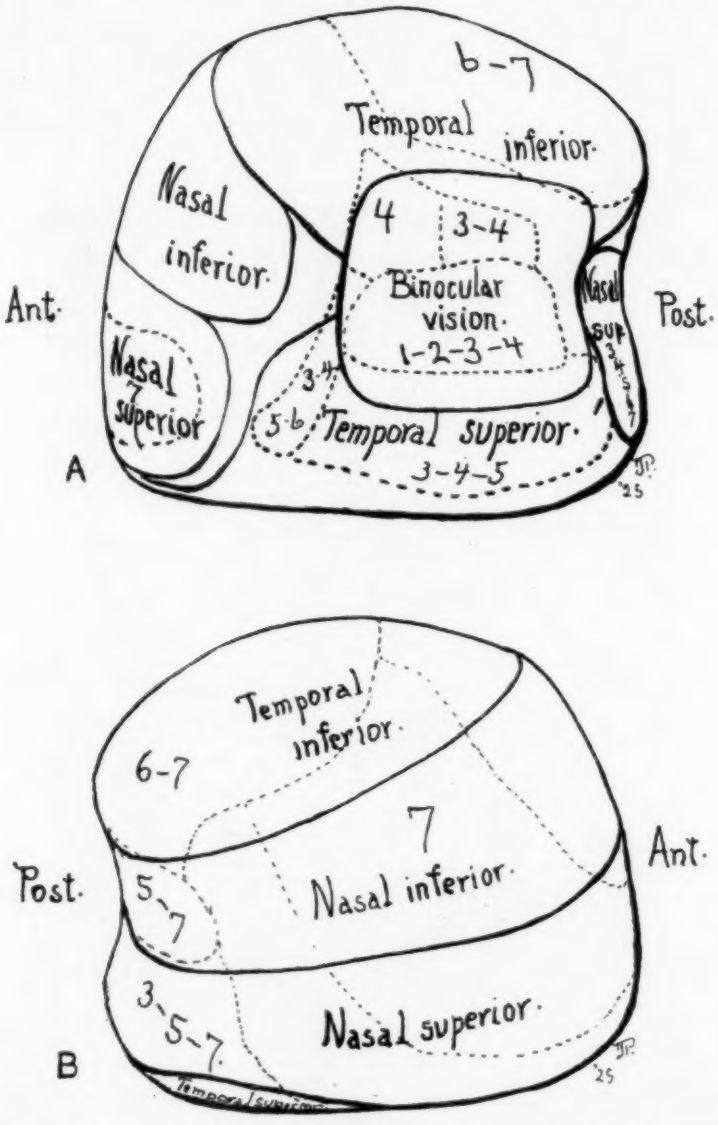


Fig. 11.—*A* is the diagram of the right corpus geniculatum of a rabbit, seen from the mesial aspect; the ventral nucleus, which does not seem to have connections with the striate cortex or retina, is not shown; the areas respectively representing the various retinal quadrants, according to Brouwer and Zeeman, are delineated with heavy continuous lines (schematically) the representation of the binocular field of vision, according to Overbosch, is similarly shown; the areas enclosed by dotted lines are those in which the degenerations seen in different experiments overlap; the numbers in these areas refer to the experiment numbers respectively; *B*, diagram of the right corpus geniculatum of the rabbit, seen from the lateral side; description and notation as in *A*.

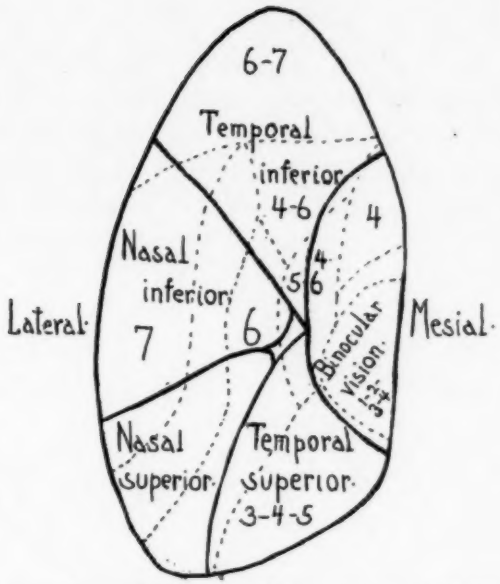


Fig. 12.—Diagram of a section through the corpus geniculatum externum of the rabbit, slightly posterior to its middle point; the representation of different portions of the retina, and the overlapping areas of atrophy, are outlined as in figure 11.

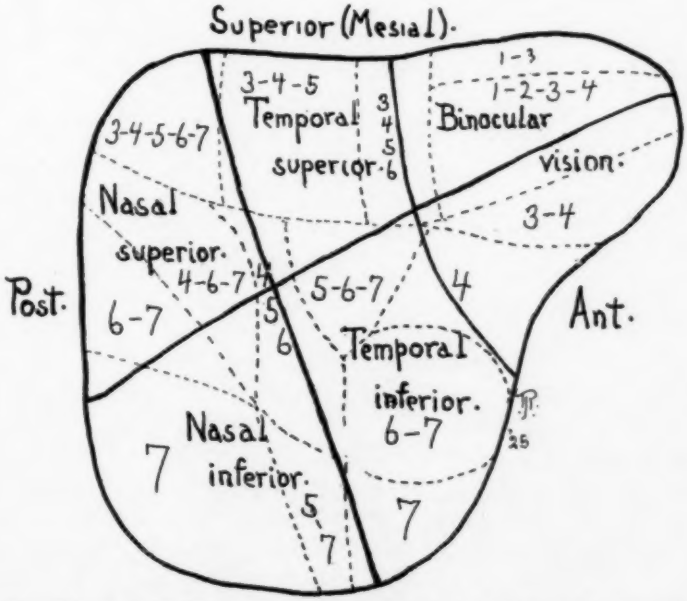


Fig. 13.—Diagram of the right striate cortex of the rabbit; the areas of overlap of the lesions produced in the respective experiments are outlined by a dotted line; the numbers refer to the experiment numbers; the probable approximate areas of the representation of the various retinal quadrants and of the field of binocular vision have been delineated by heavy lines to correspond with figures 11, *A* and *B*, and 12.

of the striate cortex, and the temporal quadrants on the antero-superior portion. The field for binocular vision has a relatively large representation anterior to that of the temporal quadrants.

5. The superior retinal quadrants are represented superiorly in the cortex, the inferior quadrants inferiorly; so that a second rotation of the field must take place in the geniculocortical neuron.

NEUROMYELO-ENCEPHALITIS DURING AND FOLLOWING AN EPIDEMIC OF HICCUP

DIVERSE LOCALIZATION OF STREPTOCOCCI *

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During the autumn and early winter of 1924, there occurred in various parts of the United States and Canada, in conjunction with relatively mild infections of the respiratory tract, an unprecedented number of cases of persistent hiccup, and cases of nausea and vomiting with or without hiccup, and of neuritis associated with or without myelitis and encephalitis. Cadham [1] describes such an epidemic in Winnipeg. Habermaas [2] reports the occurrence of a large number of cases in St. Louis. Rochester and the surrounding community had a similar outbreak. Boyd [3] mentions the occurrence, during the epidemic of encephalitis in Winnipeg in 1923, of hyperesthesia of the head, arms and body in nonencephalitic persons who were definitely ill and in persons who appeared to be well.

Opportunities to observe the course of epidemic infections are especially good in rural and small urban communities. I have observed and had accounts of epidemics that ran a characteristic course even without much evidence of contact infection. The experience related to me personally of Dr. F. W. Gessner of Dysart, Ia., a town of 1,000 persons, is especially instructive. For about four weeks, beginning the latter part of November, 1924, Dr. Gessner began to see patients with persistent hiccup. At one time he had five new patients in one day, chiefly in the country, within a radius of ten miles. In no instance could contact infection be established. Some of the patients had mild colds or sore throats, but in none was the symptom of respiratory infection severe. None vomited or was nauseated, and there was no tenderness of the scalp or other neuritic pains. During the succeeding eight weeks, infections of the upper respiratory tract became prevalent, resembling a mild

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* From the Division of Experimental Bacteriology, the Mayo Foundation.

* Read before the American Association of Pathologists and Bacteriologists, Washington, D. C., May, 1925.

1. Cadham, F. T.: Hiccup: the Winnipeg Epidemic, J. A. M. A. **84**:580-582 (Feb. 21) 1925.

2. Habermaas, A.: Ear, Nose and Throat Symposium, Weekly Bull. St. Louis M. Soc. **19**:9-10, 1925.

3. Boyd, William: Epidemic Encephalitis: the Second Winnipeg Outbreak, Quart. J. Med. **18**:153-173 (Jan.) 1925.

epidemic of influenza associated with gastro-intestinal symptoms, especially in children. Coincidently there occurred many cases of persistent nausea and vomiting, often followed by tenderness of the scalp, peculiar headaches, shooting pains into the ears, hyperesthesia and neuritic pains, sometimes associated with muscular twitchings. In most cases the symptoms were mild and lasted for only a short period, but in one case they progressed, and outspoken symptoms and signs of a neuromyelo-encephalitis developed, ending in death. In certain communities hiccup was the most common symptom; in others nausea and vomiting or deep-seated, unproductive coughing were most common; while in still others neuritic pains, often unilateral, of scalp or forehead, or outspoken neuritis dominated the picture. Marked changes in the character of the symptoms sometimes occurred during the course of the epidemic. This was true of the epidemic in Rochester. The cases of hiccup occurred chiefly between Nov. 17 and Dec. 9, 1924, and the cases of nausea and vomiting and of neuritic pains during December and the first three weeks in January. The epidemic of hiccup differed from those I had studied previously in that mild infection of the respiratory tract was more common, hiccup was not so severe and was more intermittent, and a number of patients were nauseated and vomited at the outset and had neuritic pains of the forehead or scalp as the hiccup disappeared. Spasms of the diaphragm were reproduced in this epidemic, as in those previously studied,[4] by intracerebral inoculation into rabbits of a somewhat peculiar streptococcus isolated from the nasopharynx or tonsils. Gross lesions of the peripheral nerves and spinal cord rarely occurred. As the respiratory infections became more severe the hiccup disappeared, and in its place were severe nausea, epigastric pain, persistent vomiting, lasting for from one to three days, and deep-seated spasmodic cough. These symptoms varied greatly both in severity and in situation. Most of the patients recovered without further symptoms. Others, more often in the later stages of the epidemic, developed neuritic pains, especially of the forehead or scalp, the muscles of the neck or shoulders and the external ear and, more rarely, tingling, numbness and tenderness along the nerves of the extremities. Others had neuritis and localized myositis (case 5). The attacks usually lasted for from five to ten days and then disappeared. In some cases, however, especially those showing evident foci of infection, clinical findings of neuritis involving motor fibers, and of myelitis with or without encephalitis, supervened (cases 3 and 4). Considering the striking

4. Rosenow, E. C.: Diaphragmatic Spasms in Animals Produced with a Streptococcus from Epidemic Hiccup. Preliminary Report, J. A. M. A. **76**:1745-1747 (June 18) 1921; The Production of Spasms of the Diaphragm in Animals with a streptococcus from Epidemic Hiccup, J. Infect. Dis. **32**:41-71 (Jan.) 1923.

results obtained in the cases of hiccup, the relatively mild character of the infection of the respiratory tract and the nature of metastatic disease of the nervous system, it was thought that studies similar to those made in epidemic hiccup and encephalitis might serve to isolate the causative organism from infection atria, and perhaps determine the reason for the changes in the character of the epidemic. The results obtained in the cases of hiccup will be reported elsewhere. I wish to report here results of experiments in cases of nausea and vomiting, and neuritis and allied conditions which developed during and following the epidemic of hiccup.

TECHNIC

The technic was similar to that used in a study of epidemic hiccup and encephalitis, and consisted of making cultures and animal inoculations with material obtained from nasopharyngeal and tonsillar swabbings. The swabs were thoroughly washed in 2 cc. of a 0.2 per cent gelatin Locke solution. From this, cultures and animal inoculations were made. A blood-agar plate and one or more tubes of tall columns of glucose brain broth were inoculated as a routine. The procedure in animal inoculations consisted of injecting, under ether anesthesia, 0.1 and 0.2 cc. of the suspension intracerebrally into two rabbits. If the results in the rabbits were positive, then further inoculations were made with pure cultures of the organism isolated from brain and cord or blood of these animals; if negative, the primary culture in glucose brain broth, diluted from 1:100 to 1:1,000, was injected in like amounts. Intravenous injections were made with the primary culture in glucose brain broth and with pure cultures isolated from animals in which the findings were positive. The animals were observed from three to five times a day for symptoms, and as soon after death as possible were examined in a strong light for lesions. Cultures after death were made as a routine of pipettings from brain and cord and from the blood, in some instances from other organs also and from hemorrhagic lesions in nerves. Sections of brain and cord and of nerves were made after fixation in 10 per cent solution of formaldehyde, or by Kaiserling's method. These were stained for cellular changes with hematoxylin and eosin, and for bacteria by the Gram method.

CLINICAL FINDINGS AND EXPERIMENTAL RESULTS IN INDIVIDUAL CASES

CASE 1.—A nurse, aged 30, awakened one night, six days after contracting a "cold" in the head, with intense nausea associated with repeated efforts at vomiting. Her nausea continued for two days, during which time she was unable to eat, the mere thought, sight or smell of food increasing the nausea. There was no pain, although there was a feeling of weight and fulness referable to the stomach. She also had a burning sensation of the throat which radiated into the

ears. November 27, the third day of illness, I saw the patient; she was feeling much better. The throat was hyperemic, the tonsils were absent, the ear drums were normal and the tongue was slightly coated. The leukocyte count was 10,500; the temperature and pulse were normal. Diplopia was absent; the expression of the face was normal; pressure over the points of exit of branches of the fifth nerve and over the scalp and ears failed to elicit tenderness. During convalescence, which was uneventful, the patient developed a spasmodic cough which lasted for several weeks. The head of the family of four in which she lived had had an attack of persistent hiccup ten days previously, associated with nausea and vomiting at the outset. Three weeks later a severe attack of herpes zoster developed. The mother and a boy, aged 4, developed colds about one week after the nurse became ill. The mother recovered without further symptoms; the boy had a diarrhea which lasted for ten days.

Two rabbits were injected intracerebrally with a suspension of the swabbings from the nasopharynx of the patient. The one receiving 0.1 cc. had tremor of the masseters, and a slight increase in respiration which lasted for two days. Following this, it was abnormally sleepy for about a week, and then recovered. The one receiving 0.2 cc. had a similar but more severe train of symptoms. There were severe tremor and twitchings of the masseters. Two days after the injection the animal appeared uncomfortable, was tremulous and disinclined to move and coughed spasmodically or sneezed. Later the extremities became weak, and respiration was labored and irregular. It was found dead four days after injection. Necropsy revealed moderate congestion of the vessels of the meninges, no gross lesions at the point of injection in the right cerebral lobe, moderate postmortem digestion of the stomach and no lesions of the spinal cord or peripheral nerves. Cultures of the pipetted material from the brain and spinal fluid yielded countless numbers of green-producing streptococci, and in the blood were a few colonies.

A medium-sized dog was injected intracerebrally with 0.5 cc. of the primary culture in hormone broth of the nasopharyngeal swabbing. Aside from occasional clonic spasms of the muscles of the neck the animal was well the day after the injection. On the second day it had repeated attacks of vomiting, in which only small amounts of bile-stained mucus were regurgitated. The vomiting had disappeared by the third day but the general condition was worse. The dog refused food and was disinclined to move. On the morning of the fifth day it was found dead. There were no lesions of the stomach, diaphragm or vagus and phrenic nerves. An abscess in the right cerebral hemisphere was found which yielded a pure culture of the same streptococcus isolated from the rabbits. The blood was sterile.

Comment.—This case was similar to many others of epidemic nausea and vomiting. Its close relationship to hiccup is shown by the fact that a case of hiccup had occurred a short time before in the household in which the patient lived, by the similarity of the organisms isolated and by the symptoms in the animals.

CASE 2.—The patient, a laboratory assistant, developed sore throat Jan. 14, 1924, and two days later awoke in the morning with intense nausea, a feeling of weight in the stomach and general malaise. The sore throat and nausea disappeared in two days. She had a very tender scalp for several days, followed by neuritic pains in the forearms, lasting about a week. Recovery was uneventful. January 16, the throat was moderately red; the tongue was clear; the temperature was

slightly elevated, and the pulse was slow. The suspension of nasopharyngeal swabbings contained a large number of colonies of green-producing and slightly hemolytic streptococci. The two animals injected with the suspension obtained January 16, and the one injected with the swabbing obtained January 17, developed similar trains of symptoms, well illustrated in the following protocol.

Rabbit 694.—This animal, weighing 1,320 Gm., was injected intracerebrally January 16 with 0.2 cc. of a suspension of the nasopharyngeal swabbing. January 17 and 18, respirations were increased and the head was wabbly. January 19, marked weakness of the fore extremities and muscles of the neck had developed. There were no spasms of muscles. January 20, the animal was found dead. Examination revealed moderate congestion of the vessels of the meninges, especially of the pia surrounding the cervical nerve roots, in which a number of hemorrhages were found. There were no lesions at the point of inoculation nor

Figure 1

Figure 2

Fig. 1.—Hemorrhages in superior branch of the fifth nerve near the gasserian ganglion; rabbit 694; × 2.

Fig. 2.—Hermorrhage in a cervical nerve near the point of exit between the deep muscles of the neck; rabbit 694; × 2.

gross hemorrhages of the brain or cord. The superior branch of the fifth nerve was hemorrhagic beginning 0.5 cm. peripheral to the gasserian ganglion (fig. 1), and there were a few hemorrhages in the cervical nerves (fig. 2).

Cultures from pipetted material from the brain and cord revealed large numbers of colonies of green-producing streptococci in pure form, and from the blood a few colonies. Sections revealed leukocytic infiltration surrounding vessels of the brain and cord, especially of the pia over the anterior aspect of the medulla; several small areas of hemorrhagic necrosis and leukocytic infiltration in the medulla along the floor of the fourth ventricle; hemorrhages and leukocytic infiltration of the superior branch of the fifth nerve, and Gram-staining diplococci singly and in short chains in the areas of infiltration in the medulla and fifth nerve (fig. 3, *d*). One rabbit was injected intravenously with 1 cc. of a four-day culture in hormone blood broth of the streptococcus isolated from the brain of rabbit 694, after three rapidly made subcultures. The rabbit became ataxic and weak in the fore extremities, and was chloroformed on the second day. Hemorrhagic lesions of the middle branch of the fifth nerve (fig. 4) and of the nerves

of the fore extremities were found (fig. 5), with no gross lesions elsewhere. Cultures from the blood were sterile; those from the brain yielded the streptococcus injected.

January 2, two dogs were injected intracerebrally under ether anesthesia with 1 cc. each of a five-hour culture in the third rapidly made subculture of the streptococcus isolated from the brain of rabbit 694. Both animals remained well the day of injection. The second day they were salivated, refused food, swallowed frequently and vomited repeatedly, at first partially digested food, later bile-stained mucus, after much retching. They were abnormally quiet and refused food for several days; respirations were normal, and there were no tremors or twitchings. Four days later they appeared to have recovered, and ate food and drank water normally. These two dogs, together with a normal dog, were then injected

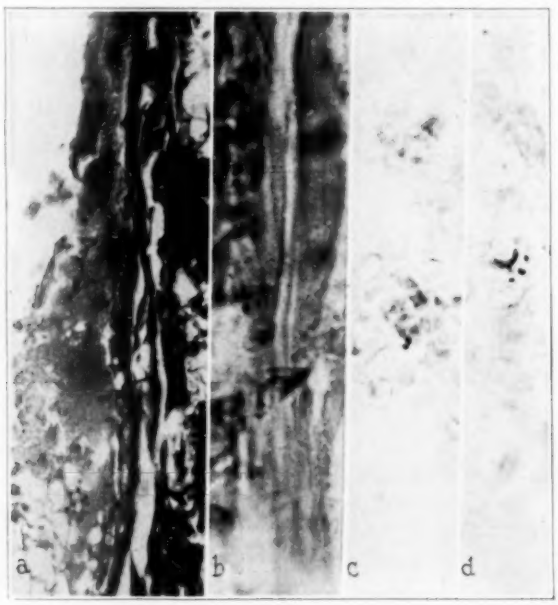

Fig. 3.—*a*, section of the branch of the fifth nerve of a dog injected intravenously with a culture of the streptococcus from case 2, after one animal passage; the hemorrhagic edema and leukocytic infiltration may be noted; hematoxylin and eosin stain; × 75. *b* and *c*, diplococci in the area shown in *a;* Gram stain; × 1,000. *d*, short chains of streptococci and diplococci in the fifth nerve of the rabbit referred to in figure 4; Gram stain; × 1,000.

intravenously with 20 cc. of the same culture after four days of incubation. One of the previously injected animals became prostrated, but did not vomit, and later developed weakness of the left foreleg. The other two vomited repeatedly between one and four hours after injection. All three recovered and were chloroformed one week later. In one no lesion was found; in the other was a small abscess in the brain at the point of injection. Cultures from the wall of the abscess revealed a pure culture of green-producing streptococci while cultures from blood and brain of all remained sterile. The dog not previously injected

revealed a hemorrhagic area in the middle branch of the fifth nerve 1 cm. distal to the ganglion. Sections revealed hemorrhagic edema and leukocytic infiltration (fig. 3, *a*), and diplococci (fig. 3, *b* and *c*).

Fig. 4.—Hemorrhage in the middle branch of the fifth nerve in a rabbit injected intravenously with the streptococcus from case 2 after one animal passage and three subcultures.

Fig. 5.—Marked hemorrhage of the nerves of the fore extremities in the rabbit referred to in figure 4.

Comment.—The clinical findings in this case were similar to those in other cases of epidemic nausea and neuritis and were almost identical with those that developed at about the same time in a laboratory assistant who worked in the same room. The findings in the animals resembled those in the patient; not only were the symptoms similar, but they were also usually transient, and the gross lesions were correspondingly few and slight and situated chiefly in nerves and medulla.

Case 3.—A middle-aged physician experienced for the first time, Nov. 27, 1924, difficulty in lifting the left foot (dorsal flexion). This continued for several days until walking was difficult, and then it rapidly improved. There were no other symptoms in the foot or leg. During this time he had had a cold with obstruction of respiration through the left nostril. The cold responded favorably to chlorine treatment. A day later there was persistent discharge from the nose which then cleared up promptly. December 22, the foot-drop had disappeared, but the patient had conjunctivitis and blepharospasm of the left eye and later of the right eye. After a few days he again had a mucopurulent nasal discharge which was somewhat streaked with blood. About this time pain and tenderness of the skin on the ulnar sides of the wrists, extending into the fourth and little fingers, appeared. The pain was never intense and at times was associated with paresthesia. There was no swelling of the joints. For nearly two weeks, beginning soon after the onset of the neuritis of the wrists and fingers, the

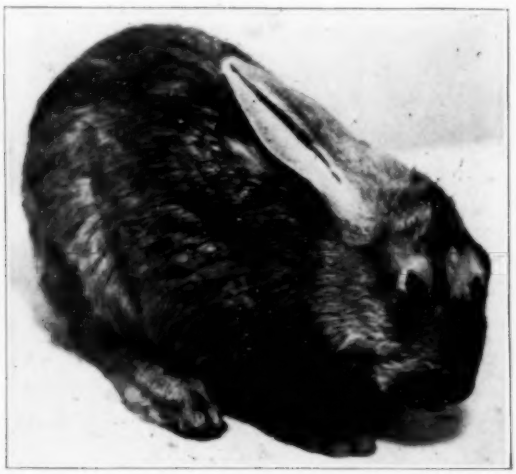

Fig. 6.—Rabbit injected intracerebrally eleven days before with a diluted suspension of the nasopharyngeal swab from case 3, and intravenously one week before with the culture after one animal passage; the marked lethargy may be noted as compared with the rabbit shown in figure 11.

patient became very sleepy; he often slept twelve or more hours at night, and while refreshed in the morning he sometimes was compelled to sleep between 5 and 6 o'clock in the afternoon. After an hour or two in bed he repeatedly had slight chills with sweating. By January 22 the abnormal drowsiness had completely disappeared, the pain and tenderness in the hands were less marked, and the patient felt much better. He then became weak; this lasted for four or five days, after which improvement was uninterrupted. Examination of the throat January 10, the day the nasopharyngeal swab was made, revealed only moderate congestion, and the sinuses were clear. Blood-agar platings of the nasopharyngeal swab showed the usual throat flora, colonies of green-producing streptococci predominating.

Four rabbits were injected intracerebrally with the suspension of the nasopharyngeal swabbing in gelatin Locke solution, two receiving 0.1 and 0.2 cc. of

the undiluted suspension, and two 0.1 and 0.2 cc., respectively, of the suspension diluted 1:100. The first two developed marked weakness of the extremities, the one receiving 0.1 cc. becoming abnormally sleepy, the other becoming abnormally excited and tremulous as ascending paralysis ensued. Both died on the second day after injection from paralysis of the respiratory center or muscles. Both revealed hemorrhagic infiltration of the right frontal lobe surrounding the point of inoculation, numerous small hemorrhages throughout the cord and hemorrhages with edema along the vertebral nerve roots and in and around the peripheral

Figure 7

Figure 8

Fig. 7.—Cord of rabbit injected intravenously twenty-four hours previously with the streptococcus from case 3, after one animal passage. The hemorrhages, especially surrounding the nerve roots, may be noted.

Fig. 8.—Hemorrhagic peripheral nerves of the rabbit referred to in figure 7.

nerves, especially those of the popliteal space and thorax. Cultures from the pipetted material, from the brain of both and the blood of one, yielded pure cultures of green-producing streptococci. Two days after the injection, the two receiving the diluted suspension developed weakness of the extremities which lasted several days; this was associated with lethargy (fig. 6) in one, and ataxia in the other. Both recovered and were injected intravenously four days later, together with two normal rabbits, with 5 cc. of the primary eighteen-hour culture in hormone brain broth of the material from the brain of one of the rabbits that

died. Three of the rabbits died within twenty-four hours, and all revealed hemorrhagic edema of the pia surrounding the nerve roots, especially marked in the cervical and sacral regions (fig. 7) and hemorrhagic lesions of the peripheral nerves (fig. 8). Two revealed hemorrhages in the tricuspid valve. Sections of the cord and nerves showed diplococci in the areas of hemorrhage and infiltration (fig. 9, *a* and *b*, and 10, *a*). The remaining rabbit was lethargic for about a week (fig. 6) after injection of the 1:100 dilution of nasopharyngeal swabbings, then became muscle-sore, protected its hind legs in hopping and lost much weight. Postmortem examination revealed a gray ventricle of stony hardness and vegetative endocarditis of the mitral valve. Cultures from the brain, cord and blood of all four rabbits revealed pure growths of the green-producing streptococcus. Sections of the brain of the rabbit that was lethargic revealed an area of round-cell infiltration in the region of the corpus striatum. The primary culture in glucose brain broth of the streptococcus isolated from one of the rabbits that showed numerous hemorrhages of the cord (second rabbit passage) was injected into three rabbits and four mice. Two rabbits and two mice received the culture diluted with equal parts of sodium chloride solution, one rabbit with 0.1 cc., intracerebrally, the other with 10 cc., intravenously. The two mice received 1 cc. each, intraperitoneally. The third rabbit received 10 cc. intravenously of equal parts of the culture and 1 per cent sodium ricinoleate in sodium chloride solution. The rabbit injected intracerebrally died from respiratory failure, due to ascending paralysis without associated myoclonus. Examination revealed the picture of a hemorrhagic pachymeningitis with hemorrhages in the cord and in the nerve trunks of the brachial plexus, and diplococci in the hemorrhagic areas (fig. 10, *b*). Cultures from the blood were sterile; those from the pipetted material from the brain contained many colonies of the streptococci. The animal injected intravenously with the broth culture diluted with sodium chloride solution died from progressive weakness the day after injection. It showed marked softening of brain and cord, hemorrhages of nerve trunks of the lower extremities and of the brachial plexus and hemorrhagic edema of the muscles of the thorax and surrounding nerves, and a left ventricle of stony hardness. Cultures from brain and blood yielded pure growths of the streptococcus. The rabbit receiving equal parts of broth culture and soap solution became extremely weak and died ten hours after the injection. Examination revealed marked congestion of the vessels in the sheaths of the nerves of the lateral aspect of the thorax, forearms and legs, marked hemorrhages of nerves for a distance of 1 cm. at the lower and inner aspect of the legs and marked postmortem digestion of the stomach. Cultures of the suspension injected and those from brain and blood after death remained sterile.

Comment.—The points of particular interest in this case are the evidence of widespread lesions in the nervous system, including sensory and motor nerves, cord and brain, the production of similar symptoms in animals, and the pronounced lesions in nerve, cord and brain following both intracerebral and intravenous inoculation of the streptococcus isolated from the throat of the patient.

CASE 4.—A man, aged 58, came to the clinic Nov. 4, 1924. Five days previously, after a severe chilling, he had developed a cold, with severe generalized pain in the muscles, including backache. He was sent to the hospital for observation and study. On admission he complained of pains in the thighs, particularly along the course of the sciatic nerves, and in the calf of the legs, making walking

difficult. His general condition was good, but there was marked tenderness over the sciatic nerves and muscles of the legs. He had suffered for years with backaches due to hypertrophic arthritis of the spine.

December 7 he became nauseated. December 8 he complained of marked soreness of the toes, and the muscles of the legs and the nerve trunks were very sensitive to pressure. The leukocyte count was 5,700. December 9 he was still nauseated. The cerebrospinal fluid was under 10 cm. pressure, and contained three small lymphocytes. The Kolmer test of the blood and spinal fluid was negative. December 12, he had developed incontinence of urine and feces, weakness and numbness of the legs and lower part of the body extending to the level

Fig. 9.—Sections of peripheral nerve in the rabbit injected intravenously with the streptococcus from case 3 after one animal passage. *a*, the marked hemorrhage within the nerve substance may be noted; hematoxylin and eosin stain; × 75. *b*, the diplococci at the junction of the nerve fiber and hemorrhage may be noted; Gram stain; × 1,000.

of the umbilicus, pain and numbness of the upper extremities extending to the elbows and difficulty in swallowing, especially solid foods. His condition did not improve while he was in the hospital and he went home. He returned May 3, 1925, at which time he complained of pain and tenderness of the soles of the feet, pain in the shoulders, general weakness and distress in the stomach after meals; the difficulty in swallowing had entirely disappeared. Examination was largely negative although tenderness along the sciatic nerves was still present.

Suspensions of the pus from the tonsils and of the nasopharyngeal swab were each injected intracerebrally into two rabbits in the usual manner. There developed marked difficulty in breathing and rapidly increasing weakness asso-

ciated with ataxia; two had mild spasms of the abdominal muscles. They died within two days. All three had gross hemorrhages in the cord; two had hemorrhages in nerves as well. Cultures from the brain and cord and blood yielded a pure growth of the streptococcus. Sections of the brain revealed hemorrhages, leukocytic infiltration and diplococci, especially surrounding the nerve roots (fig. 10, *c*). The fourth rabbit developed moderate hyperpnea, slight ataxia, disinclination to hop, general weakness and loss in weight during the course of a week; then it died. No gross lesion of brain or cord could be found, and cultures were negative, but sections revealed marked perivascular and parenchymatous round-cell infiltration in the brain and medulla. Two rabbits were injected with a mixture of the cultures in glucose brain broth of the brains of the three rabbits

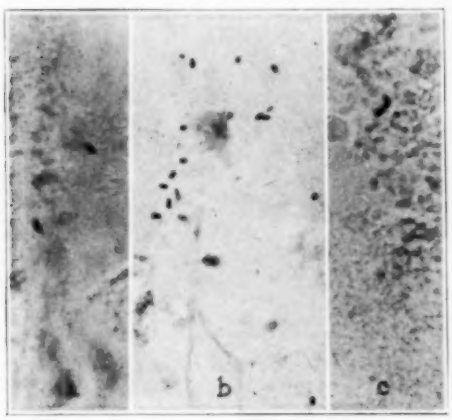

Figure 10

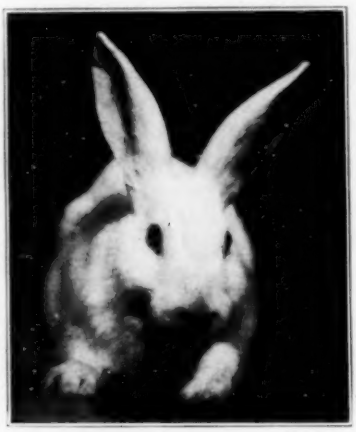

Figure 11

Fig. 10.—Diplococci in lesions of rabbits. *a*, hemorrhagic area in the nerves shown in figure 8; *b*, hemorrhagic lesions of the rabbit injected intracerebrally with streptococci from case 3 after two animal passages; *c*, area of leukocytic infiltrations surrounding nerve roots and cord of rabbit injected intracerebrally with the streptococcus from case 5. Gram stain; × 1,000.

Fig. 11.—Rabbit injected intravenously on three occasions with the streptococcus from case 4 in the second and third animal passages. The drop-foot on the left side may be noted.

that died soon after injection. One received 0.2 cc. of a 1:1,000 dilution, the other 0.2 cc. of a 1:100,000 dilution. Within twenty-four hours the former developed moderate hyperpnea, weakness of the fore extremities and ataxia, which disappeared in a few days. At the end of a week the animal had lost much weight; the muscles of the extremities had become markedly atrophied, and peculiar movements of the head, resembling spasmodic torticollis, had developed. The second rabbit became very weak and dyspneic, and died from what appeared to be a choking attack. Necropsy revealed a large hemorrhage in the right cerebral ventricle and marked congestion along the cervical nerve roots. The fresh filtrate of nasopharyngeal washings, obtained December 10, was injected intracerebrally into two rabbits, one receiving 1 cc., the other 1.5 cc. Within fifteen minutes both had developed marked hyperpnea, moderate weakness and generalized tremor

and twitchings of skeletal muscles, but no spasms of the diaphragm. These symptoms continued for about half an hour and then disappeared. Four rabbits were injected intravenously with the streptococcus in the second and third animal passage. Each received 5 cc. of the glucose-brain-broth culture on three occasions two days apart. One had difficulty in extending its left forepaw (fig. 11); one died from intercurrent *Bacillus bronchisepticus* pneumonia, and two lost markedly in weight without showing evidence of neuritis or myelitis.

Comment.—The points of special interest in this case are the evidence of widespread involvement of nerves, spinal cord and medulla, and the occurrence of lesions in these structures in the animals inoculated.

Fig. 12.—Hemorrhages in the muscles of the neck surrounding points of exit of cervical nerves in the rabbit injected intravenously with the streptococcus from case 5 after one animal passage.

CASE 5.—A young woman, stenographer by occupation, during a severe attack of cold in January, 1924, had pain over the right eye, tenderness of the forehead and a severe steady aching pain in the muscles of the neck. Acetylsalicylic acid and the application of heat relieved her temporarily, but she gradually grew worse and went home January 12 with a temperature of 102. She remained in bed for two days, the temperature ranging from 101 to 102. The pain in the neck continued until January 19, the patient taking salicylates meanwhile for relief. January 10, when a nasopharyngeal swab was taken, the throat was found to be hyperemic, and the tonsils badly infected. The cervical glands were enlarged and tender. Deep pressure over and between the muscles of the neck was painful, but superficial tenderness was not elicited. January 24, the tonsils were removed,

following which there was pain through the entire body lasting for five days, associated with transient hiccup. Recovery thereafter was rapid and complete.

One rabbit was injected intracerebrally with 0.1 cc. of the primary culture in hormone brain broth of a nasopharyngeal swab obtained January 10. The next day respiration was greatly increased as the animal developed marked weakness and rhythmic spasms of the diaphragm and abdominal muscles. It was chloroformed. Hemorrhages were found throughout the length of the cord; hemorrhagic edema surrounded the nerve roots, and there were a few hemorrhagic areas in the muscles of the thorax along the nerves. Cultures of the brain yielded countless colonies of green-producing streptococci in pure form; those of the blood were sterile. The primary culture in hormone brain broth from this rabbit was injected intravenously (3 cc.) into another rabbit; it was found dead the following day. Examination revealed hemorrhagic edema of the muscles surrounding the nerve trunks in the anterior cervical region (fig. 12), in the region

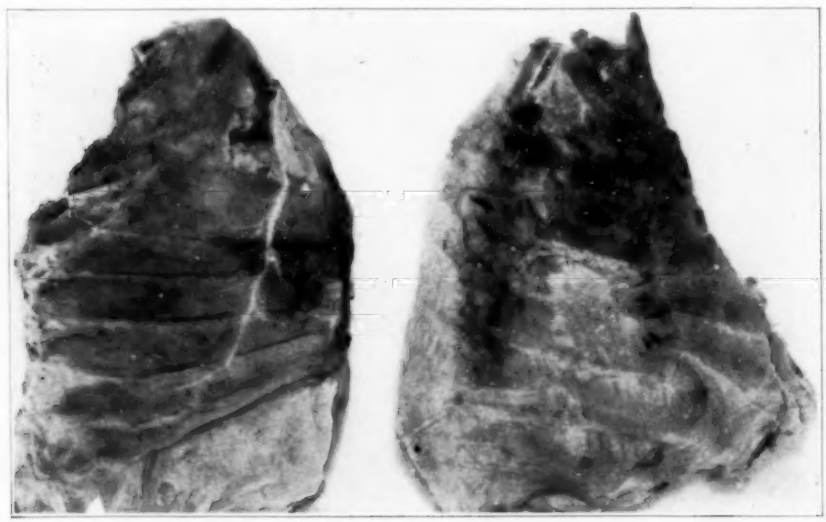

Fig. 13.—Hemorrhages of muscles surrounding points of exit of nerves of the thorax of rabbit referred to in figure 2.

over the right thorax (fig. 13), in that over the lateral and outer aspect of the right leg and of the cervical nerve roots; and hemorrhagic infiltration of the deep muscles over the anterior aspect of the neck, over the lateral aspect of the thorax, usually of the surrounding nerves, and over the upper and outer aspect of both legs, and localized hemorrhages in the myocardium. Cultures from the brain and blood yielded pure growths of the streptococcus. A single colony from the brain of the first rabbit was inoculated into two tubes of glucose broth. The one was incubated aërobically., the other anaërobically for two days, when both were filtered. The filtrate of each (1.5 cc.) was injected intracerebrally in two rabbits. The aërobic filtrate had no apparent effect. One of the two rabbits injected with the anaërobic filtrate died in four hours; the other had increased respiration for several hours and then recovered. The three rabbits that survived were then injected intravenously with 7 cc. of the corresponding anërobic filtrate. They were chloroformed two days later. All had small hemorrhagic lesions of the muscles in the lumbar or

cervical region, chiefly surrounding nerves. Cultures from the blood and lesions in muscles remained sterile. The primary culture of the streptococcus isolated from the second rabbit (second rabbit passage) was injected intravenously into two rabbits, and the sixteenth subculture into one rabbit (7 cc). One of the former failed to develop lesions. The other two had marked lesions of nerves and muscles, especially in the cervical and lumbar regions, and the one injected with the primary culture also had lesions of the myocardium and heart valves.

Comment.—The findings in this case were closely simulated in the animals; that is, they had lesions, chiefly of nerves and muscles, without gross hemorrhages in the spinal cord.

SUMMARY AND COMMENT

With a few exceptions, the findings in the animals paralleled in important respects those in the patients studied. While tremors and spasms of muscles, chiefly of jaws, ears and neck, occurred in nine of twenty-one rabbits injected intracerebrally, spasms of the diaphragm occurred in only three. Two of these were injected with the streptococcus from the two patients who had transient hiccup, and the other with the streptococcus from the patient who developed nausea and vomiting in the family in which one member had had a severe attack of hiccup a short time previously. All of the four dogs injected with the material from the two patients who were severely nauseated and vomited developed anorexia and vomited repeatedly. Only one rabbit of thirty-two (3 per cent) injected with material from four of the patients in whom lethargy was absent became lethargic, while three of twelve rabbits (25 per cent) injected with material from the patient who had lethargy manifested this symptom.

Evidence of neuritis was present in the five cases studied; correspondingly hemorrhagic lesions of nerves occurred in eight of twenty-one rabbits injected intracerebrally and ten of seventeen injected intravenously. Parallelism between findings in patients and in animals is further shown by the fact that, of sixteen rabbits injected intracerebrally with the streptococcus from the three cases in which there was no evidence of myelitis, only one (6 per cent) had gross hemorrhages of the cord or medulla, while of twenty-two injected with material from the two patients who had undoubted symptoms of myelitis, nine (43 per cent) had gross hemorrhages in the cord. Moreover, of seventy-three rabbits injected intracerebrally with material from twenty-one cases of epidemic hiccup in which search for hemorrhages in nerves and cord was made, only two revealed gross hemorrhages of peripheral nerves, four of nerve roots and six of spinal cord. In a few instances lesions containing the streptococcus were found in the stomach or colon following intracerebral inoculation, especially of strains injected after one or two animal passages, a finding considered in harmony with the fact that gastro-intestinal symptoms were not uncommon in these epidemics.

The character of the microscopic changes in the brain and cord in the animals injected in this series of cases was similar to that found in the experiments in epidemic hiccup [4] and encephalitis.[5] Hemorrhage, edema and leukocytic infiltration dominated the picture in the experiments of short duration, while later infiltration by round cells became predominant. The demonstration of organisms in the acute lesions was relatively easy; in the chronic lesions it was difficult and sometimes impossible. In animals injected with strains from cases that showed mild and transient symptoms, the microscopic as well as the gross findings were, with few exceptions, less pronounced, and the organisms in the lesions less numerous (fig. 3) than they were in animals injected with the strains from more severe cases (figs. 9 and 10), a finding in agreement with the less frequent isolation of bacteria and the lower mortality rate in the former group of animals. The organism was demonstrated to the exclusion of other forms in the lesions remote from the point of injection, including those in animals in which cultures of the blood were sterile and which were injected even with the mixture of bacteria contained in nasopharyngeal swabbings. Lesions situated similarly to those in patients were found following inoculation of nasopharyngeal swabbings, pure cultures of the living streptococcus after one or more animal passages and repeated subcultures (as high as sixteen), the dead streptococcus, filtrates of nasopharyngeal washings and filtrates of anaërobic cultures. The streptococcus isolated in these cases is morphologically and culturally indistinguishable from that in epidemic hiccup and encephalitis. Like the latter, it is of relatively low general virulence. It is a slightly elongated pleomorphic diplococcus occurring singly and in short chains. It is gram-positive, nonencapsulated and bile insoluble, and on blood agar produces small, dry, slightly elevated, nonadherent colonies surrounded by a green halo. All of the five strains were agglutinated by my polio-encephalitis immune serums, in dilutions as high as 1:100, and not by other similarly prepared immune serums. Positive precipitin reactions were obtained in the polio-encephalitis serums with the cleared suspensions of nasopharyngeal swabbings in three of four cases. The requirements for causal relationship of this streptococcus to the findings in the patients studied appeared fulfilled. Since changes in localizing power were noted in some of these strains following artificial cultivation and animal passage, the conclusion seems warranted that the changes in the character of the epidemic, from hiccup to other manifestations of a neuromyelo-encephalitis, were due to change in the tropism or localizing power of the streptococcus isolated.

5. Rosenow, E. C.: Streptococci in Relation to the Etiology of Epidemic Encephalitis: Experimental Results in Eighty-One Cases, J. Infect. Dis. **34**:329-389 (April) 1924.

GENERAL PARALYSIS TREATED WITH TRYPARSAMIDE

A CLINICOPATHOLOGIC REPORT OF A CASE *

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The mere fact that the methods of treatment of general paralysis are so numerous and changeable denotes how unsatisfactory and unsettled they are. The great hopes aroused by arsphenamine and neo-arsphenamine preparations did not materialize, and new methods have been sought, of which two are at present commanding the attention of clinicians. These are tryparsamide and inoculations with malaria or relapsing fever. Enthusiastic as are the reports on the results with tryparsamide, they are much more so with malaria. The literature on both is already too extensive even for a brief review, and we shall therefore confine ourselves to the pathologic phase of this problem. Here we might point out that while a number of reports exist dealing with the pathologic changes obtaining in cases of general paralysis treated with malaria, none is available as to the conditions in cases treated with tryparsamide. As tryparsamide had been used rather intensively in the case here recorded, it will be of interest to contrast the histopathologic findings in the brain with those in brains from patients untreated or treated with malaria inoculations.

REPORT OF CASE

Clinical History.—A man, aged 51, admitted to the Presbyterian Hospital, Jan. 10, 1922, had shown some decline in his mental capacity for over a year, and in December, 1920, had given up his position as foreman of a shop because he no longer felt equal to it. He continued working, however. In May, 1921, it was noted that he stumbled frequently and felt weak in his knees. There were occasional convulsive movements, chiefly in the left leg. His memory was failing during this year and his weight declined from 145 pounds (65.8 Kg.) in May, 1921, to 113 (51.3 Kg.) in October. In November he had consulted a physician who found that the Wassermann test with his blood was strongly positive, and he received seven injections of some kind, after which he felt stronger. He stated that he had had a chancre at the age of 24.

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* Presented before the Chicago Neurological Society, Jan. 21, 1926.

* From the pathology laboratories of the Research and Educational Hospitals of the University of Illinois College of Medicine, and the neurologic service of the Presbyterian Hospital, Chicago.

Examination.—On examination the patient was found to be rather emaciated, weighing only 107 pounds (48.5 Kg.). He was good natured, garrulous and greeted all comers as old friends. There was no slurring of speech, and he repeated the usual test sentences well. Calculation was rather poor. He said that 9 times 9 were 99 and could not subtract $1.28 from $5.00, but he subtracted $6.63 from $10.00 correctly. At times he would cry and appear frightened. The pupils were small and irregular and reacted in accommodation but not to light. All tendon reflexes were present except the right ankle jerk. There was a slight Romberg's sign. There were occasional choreiform movements in the left leg and a coarse tremor in the left hand. The condition of the blood and spinal fluid is seen in the table, in which the treatment administered is also recorded.

Serologic Findings, Treatment and Course

Date	Wassermann Reaction: Blood	Wassermann Reaction: Fluid	Cells	Ross-Jones Reaction	Colloidal Gold Reaction	Intravenous Injections, Neo-arsphenamine	Intraspinal Serum Injections, Cc.
1/10/22	+++	++++	25	+	5544321100	...	..
1/17/22 to 1/30/22	.....	.....	..	....	.....	4 inj. 0.6-0.75	..
2/ 4/22	.....	.....	..	....	.....	0.9	32
2/11/22	.....	++++	25	++	5544432100	0.9	35
2/18/22	.....	.....	..	....	.....	0.9	27
2/27/22	.....	.....	..	....	.....	0.9	40
Better. Gained 19 pounds in weight							
3/16/22	.....	.....	..	....	.....	0.9	37
3/30/22 Back at work	.....	.....	..	....	.....	0.9	40
4/13/22	.....	+++	19	+	1283221100	0.9	40
5/10/22 Fainted after injection	.....	.....	..	....	.....	0.9	None
6/13/22	.....	+++	18	+	1223211000	0.9	40
8/ 3/22	.....	+++	15	+	1223211100	0.9	35
Has gained 52 pounds since January, 1922							
1/10/23	—	++++	30	+	4433210000	0.9	30
Speech slurred. Unable to work							
2/14/23	.....	.....	..	....	.....	0.75	35
3/28/23	++++	+++	1	+	4543200000	0.75	45
8/ 1/23	.....	++++	8	+	0221000000	0.75	25
8/ 9/23	.....	.....	..	....	.....	0.75	35
8/22/23 Worse mentally. Untidy	.....	.....	..	....	.....	0.75	37
Sept. and Oct. 8 tryparsamide injections							
1924 Jan. and Feb. 8 tryparsamide injections							
1/ 9/24	—	—	1	—	0121000000	...	..
8 tryparsamide injections							
5/21/24	—	—	18	+	1133210000	...	..
8 tryparsamide injections							
10/ 8/24	—	++	4	+	0110000000	...	..
1 tryparsamide injection							
1/25/25	—	—	33	+	1421100000	...	..
4/ 9/25 Died of bronchopneumonia							

Course.—When the patient was discharged, on March 1, he was decidedly improved both mentally and physically and had gained 19 pounds (8.6 Kg.) in weight from January 27 to February 17. He continued to improve and returned to work in March. On June 12 his weight was 157 pounds (71.2 Kg.), a gain of 50 pounds (22.7 Kg.) since the beginning of treatment, and he remained at work. He was at his best after the treatment in August, 1922, and did not return to the hospital until Jan. 9, 1923. He then had been unable to work for six weeks on account of general weakness and shakiness, and during this time his speech had become slurred. There was more tremor and all tendon reflexes were increased. While in the hospital he had confused spells, during which he was quite irrational. He improved considerably and was in much better condition on his return in

February, except that the speech was still slurred. He did a little work again during the spring and summer and returned in a weak condition on July 31, when his weight was 136 pounds (61.7 Kg.). He was irrational at times and had occasional involuntary evacuations. It will be seen from the accompanying table that tryparsamide treatment was started in September, 1923. When discharged on October 9, he was decidedly improved. During the following months treatment was continued and he returned to work, though he gave it up after a few weeks. When he was reexamined in May, 1924, calculation was fairly good but there was unmistakable deterioration. The tendon reflexes were considerably increased.

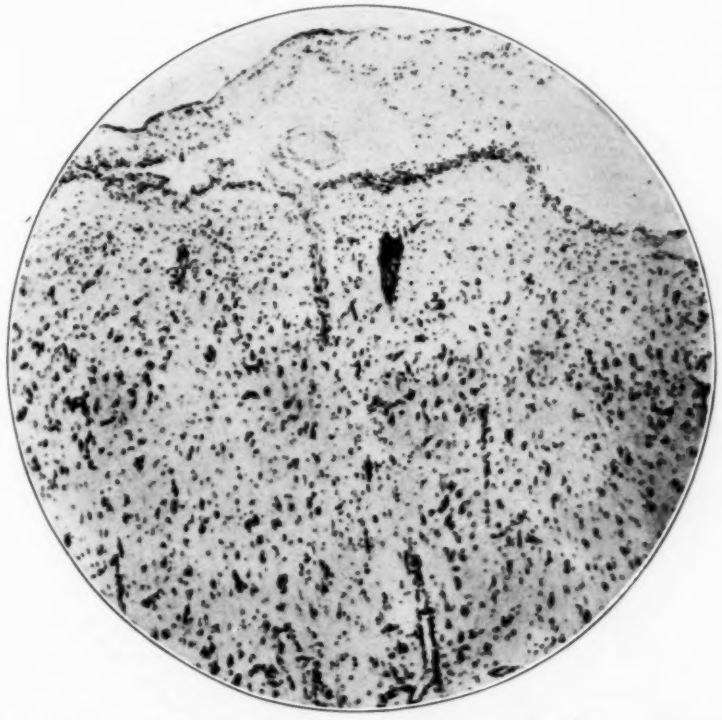

Fig. 1.—Frontal lobe: mild infiltration of the pia-arachnoid; the pial blood vessels are very slightly infiltrated, but the cortex shows a large number of blood vessels, some of which are quite densely infiltrated. Comparison should be made with figure 8. Toluidin blue × 60.

This was still more apparent in October, and at this time there was moderate concentric contraction of the visual fields and slight pallor of the optic disks. In January, 1925, when he was admitted to the hospital for the last time, the mental confusion was so marked that he was unable to give any coherent history. Nevertheless, as will be noted from the table, the Wassermann test was negative with both blood and spinal fluid. No further treatment was given. The patient gradually declined both physically and mentally. For ten days before death he had considerable fever and symptoms of a bronchial infection. He died April 9, 1925, and necropsy was performed by Dr. C. W. Apfelbach a few hours after death.

Necropsy Findings.—The skull was adherent to the dura. There were fibrous areas in the leptomeninges and in places the latter were raised by accumulations of clear fluid. There was marked atrophy of the convolutions and the cerebral vessels showed considerable arteriosclerosis.

Microscopic Findings.—There was a mild leptomeningitis (fig. 1) over the convexity and base of the brain; infiltration cells and small and large lymphocytes mixed with mesothelial and some plasma cells were scattered within the distended meshes of the pia-arachnoid and around hyperemic blood vessels and capillaries; excessive vascularization of the cortex was present with perivascular plasma cell infiltration of the capillaries and lymphocytic infiltration of the larger blood vessels; there were numerous new formed capillaries; hypertrophy of the

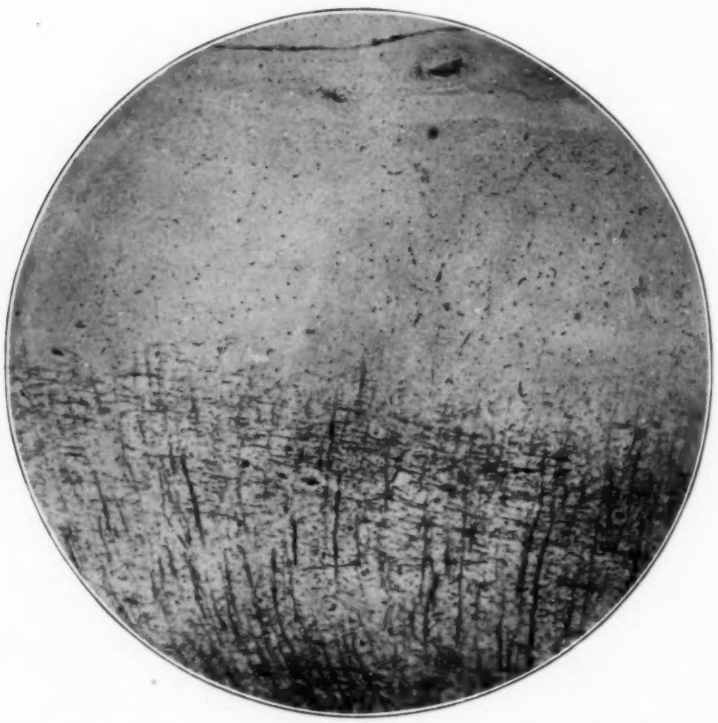

Fig. 2.—Frontal lobe: complete loss of tangential nerve fibers. Weigert-Pal × 36.

endothelial and hyperplasia of the adventitial cells existed; various forms of cell degeneration were found with phenomena of satellitosis and neuronophagia; excessive glia proliferation was present with numerous rod cells throughout the cortex; lack of tangential fibers (fig. 2) and vast accumulation of lipoid substances in the ganglion cells and adventitial spaces of the blood vessels were observed. The foregoing changes were diffuse, but were especially pronounced in the frontal and temporal lobes and in the basal ganglia, while in the occipital lobe they were rather insignificant. The staining methods of Ramón y Cajal (gold—mercuric chloride) and Del Rio Hortega (silver carbonate) revealed an enormous amount of fibrous glia (macroglia) and microglia. The former had a large cell body enclosing an eccentric nucleus and possessing numerous well devel-

Fig. 3.—Frontal cortex: excessive vascularization with an abundance of macroglia cells. Compare with figure 7. Gold-mercuric chloride stain of Cajal × 170.

Fig. 4.—Ammon's horn: numerous microglia cells of Del Rio Hortega are scattered over the visual field. Hortega's silver carbonate stain × 260.

oped, thick, ramifying processes reaching and often enveloping the capillaries and blood vessels (fig. 3). The microglia appeared entirely different. The nucleus was slightly oval, often rod shaped, and much smaller than in the macroglia; the cytoplasm was usually scanty (fig. 4), often hypertrophied with processes not only numerous but greatly increased in size, and showing an abundance of small side branches (spines). In some instances the hypertrophied processes of microglia cells lay parallel to, and as if surrounding or enveloping, the ganglion cells, which generally appeared much paler than the microglia, their cytoplasm homogeneous and the dendrites faint. As a rule they were so inconspicuous that some visual

Fig. 5.—Frontal lobe: the visual field is covered with numerous mesodermal elements—blood vessels and microglia—also macroglia; some structures such as microglia and ganglion cells may be seen with the help of a hand lens. Hortega stain × 130.

fields (Ammon's horn, frontal lobes) appeared as if made up of blood vessels, microglia and cytoplasmic glia cells (fig. 5).

Numerous blocks from the cortex were studied with the methods of Levaditi and Jahnel for the presence of spirochetes. Only with Jahnel's method did we succeed in finding them (in the left frontal lobe). They were scattered mostly singly (fig. 6), or as minute dark granules, apparently unmolested by the glia, blood vessels or microglia, that is to say their presence was not accompanied by neighborhood reactive phenomena, otherwise so abundant in the cortex and the basal ganglia. In general, the histopathologic findings may be summed up as a meningo-encephalitis typical of general paralysis.

COMMENTS

Contrasted with the condition of a normal brain, the changes outlined are decidedly striking. This is true not only of the inflammatory and the degenerative phenomena, but also of the state of the macroglia (fig. 7) and microglia. Both markedly developed, they did not appear hypertrophied or hyperplastic in a control, normal brain; in short, they were less abundant than in the case of general paralysis treated with tryparsamide.

On the other hand, an untreated case showed the same pathologic phenomena as were found in the case treated. The same mesodermal and

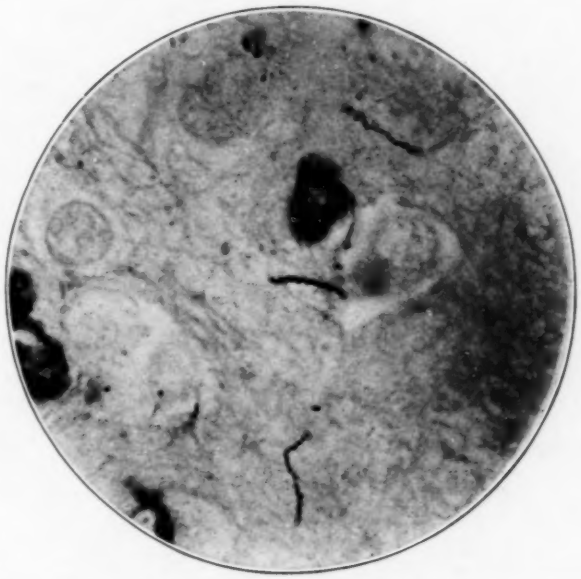

Fig. 6.—Spirochetes in the frontal lobe with absence of reactive phenomena. Jahnel's stain × 1,200.

ectodermal reactions were in evidence, the former, decidedly, much more marked. Thus, the pia, as figure 8 shows, is much more infiltrated, and the blood vessels of the cortex exhibit adventitial spaces markedly packed with lymphocytes and plasma cells which were also abundantly scattered over the parenchyma; the capillaries were more numerous and greatly proliferated. However, as pointed out elsewhere,[1] inflammatory phenomena, however severe they may be, do not possess the significance or importance of the degenerative phenomena, though both are caused by the same infection. In other words, the comparatively mild inflam-

1. Hassin, G. B., and Bassoe, Peter: Parkinsonian States: Clinico-Pathologic Studies, Arch. Neurol. & Psychiat. **15**:218 (Feb.) 1926.

matory reaction in our case does not denote that the morbid condition itself was influenced by the treatment and had therefore come to a standstill, for neither the degenerative phenomena nor the spirochetes were favorably affected by the treatment. It is thus evident that the latter did not accomplish striking results histologically. Like many other antisyphilitic drugs, tryparsamide is able neither to restore function to central nerve elements that are partly or completely damaged, nor to effect a complete destruction of the spirochetes. It may favorably

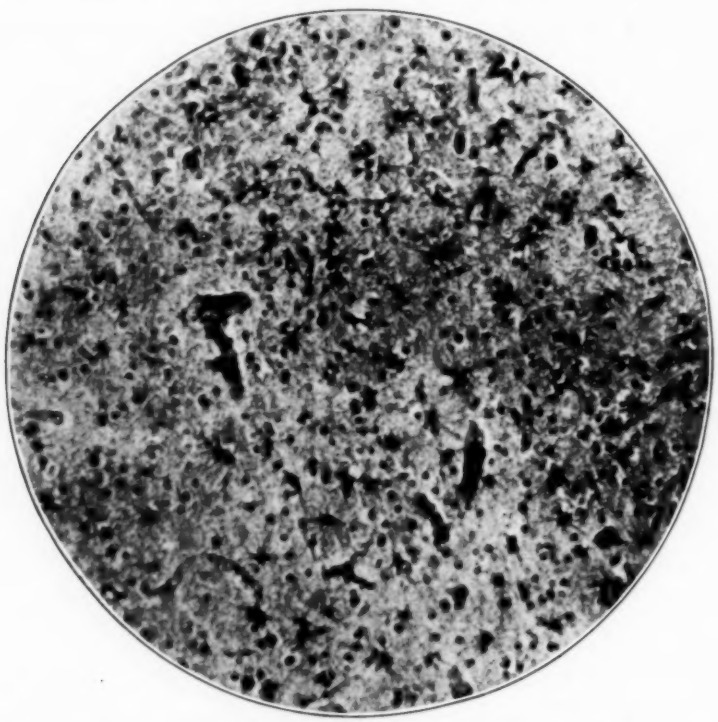

Fig. 7.—Cortex from a normal brain—macroglia is here less abundant than in general paralysis. Comparison should be made with figure 3. Stain and magnification same as in figure 3.

influence the mesodermal reactive phenomena which, however, are not as widespread, stable and irreparable, as are the degenerative phenomena. The latter are therefore more dangerous, for, once started, they progress and never stop, resembling similar conditions in multiple sclerosis, amyotrophic lateral sclerosis, subacute cord degeneration and postencephalitic states.

As we were not in a position to study the changes in cases of general paralysis treated with malaria inoculations we must refer to the con-

clusions arrived at by others. Sträussler and Koskinas,[2] who studied histologically thirty-eight cases of general paralysis treated with malaria, state that the inflammatory changes were in general mild, as in our case. They are inclined to believe that the remissions observed during and after the treatment coincided with the anatomic changes, and that the malaria treatment was responsible for both the modified anatomic changes and the remissions. According to these investigators, malaria produces first intensive hyperemia with an increase in the inflammatory

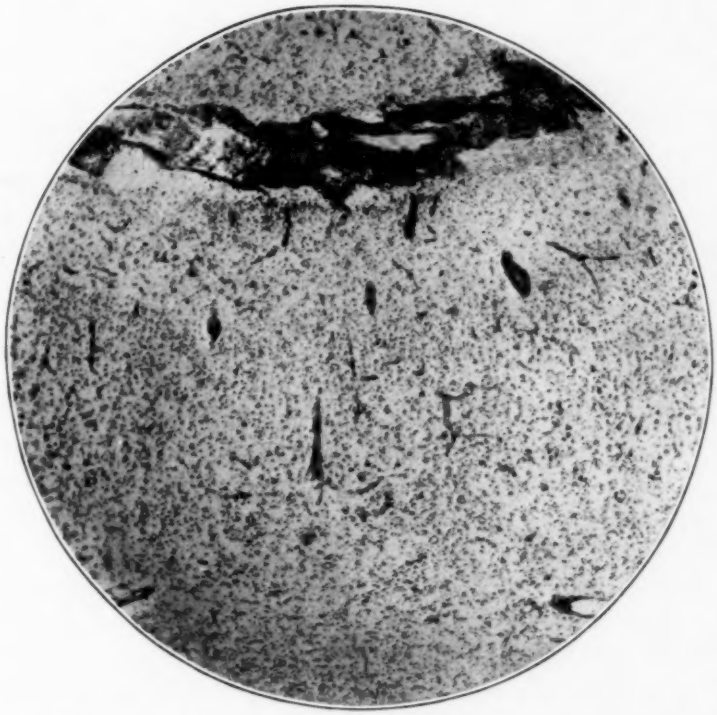

Fig. 8.—Frontal lobe from an untreated case of general paralysis: the pia is enormously infiltrated; the blood vessels are numerous and greatly infiltrated. Comparison should be made with figure 1.

phenomena, which afterward change their type—from diffuse and unspecific they become specific and granulomatous or gummatous. The powers of the organism in its fight against the infection are stimulated, leading to the transformation of the malignant brain syphilis into a benign form. These hypothetical considerations are in full accord with

2. Sträussler, E., and Koskinas, G.: Weitere Untersuchungen über den Einfluss der Malariabehandlung der progressiven Paralyse auf den histopathologischen Prozess, Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:176, 1925.

the views of A. Jakob,[3] who looks on general paralysis as a malignant syphilis of the brain. Spielmeyer [4] attacked the validity of this teaching. He justly pointed out that the inflammatory manifestations, however diffuse or intense, do not denote the case as grave clinically, and that the clinical picture does not always correspond with the actual pathologic condition; a mild clinical picture may be associated with a grave inflammatory state, and vice versa. Therefore, one is not justified in asserting that the mild inflammatory phenomena found in a brain treated with malaria or, as in our instance, with tryparsamide, signify that improvement took place clinically and was due to the treatment.

Optimistic as Sträussler and Koskinas are, they do not speak of cures, complete or incomplete, but of more or less prolonged remissions. These, however, may or may not be due to the method of treatment used, for they may be spontaneous; however prolonged they may be they cannot lead to recovery. The sole rational treatment of general paralysis is prophylactic, in the form of intensive periodic treatment of the syphilis in its early stages, controlled by periodic serologic examinations. Important as these latter are, they should not play the principal rôle in directing treatment, for there is no parallelism between the serologic reaction and the clinical picture. The latter should always be considered with the greatest care and the treatment of each case be strongly individualized.

CONCLUSIONS

1. A case of general paralysis of the insane intensely treated with neo-arsphenamine and tryparsamide showed marked improvement serologically and clinically.

2. The inflammatory, mesodermal phenomena were found to be milder than in an average case of general paralysis; they were thus influenced by the treatment.

3. The ectodermal elements, ganglion and glia cells, were not affected by the treatment.

4. Neither did the treatment effect the disappearance of the spirochetes.

5. Cure of general paralysis with modern methods—tryparsamide or malaria—cannot be expected.

6. The sole treatment of this disease is prophylactic, that is, it should be directed against syphilis in its early stages before irreparable degenerative lesions of the parenchyma set in.

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3. Jakob, A.: Ueber Entzündungsherde u. miliäre Gummen im Grosshirn bei Paralyse, Ztschr. f. d. ges. Neurol. u. Psychiat. **51**:7, 1919.

4. Spielmeyer, W.: Ueber Versuche d. anat. Paralyseforschung zur Lösung klinischer u. grundsätzlicher Fragen, Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:287, 1925.

DISCUSSION

Dr. H. Douglas Singer: The question is whether this study was made during a remission. The clinical course appears to have been progressive. I wonder whether the enormous increase of glia is not, perhaps, proportional to the length of time the condition had lasted. It is rather unusual to have the disease continue so long.

Dr. A. B. Yudelson: Does Dr. Bassoe believe the improvement is due to the Swift-Ellis treatment, and does he attach importance to this in preference to anything else?

Dr. Bassoe: There were two definite periods of remission. The first was attributed to the Swift-Ellis treatment. The patient then went back to work for several months. He slipped, and the Swift-Ellis treatment had no effect; but after his first course of tryparsamide he had the second remission and was again able to work for several months, until the final collapse came.

Dr. Hassin: It should be admitted that the remissions were of small benefit to the patient for, notwithstanding the intense and conscientious treatment with a large amount of tryparsamide, the condition became hopeless. In general paralysis the structural parenchymatous changes are evidently so far advanced that no amount of arsenic can influence them. The only favorable changes are to be found in the meninges, resembling that which occurs after treatment with malaria inoculations.

BLOOD CALCIUM AND PHOSPHORUS IN PERSONALITY DISORDERS

THE EFFECT OF ULTRAVIOLET RADIATION

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For many years there has been a popular belief that calcium and phosphorus metabolism was somewhat directly associated with the function of the nervous system. Textbooks [1] on clinical diagnosis have reported observations of decreased phosphoric acid and increased calcium content of the urine in cases of "neurasthenia," and a diminution in the relative amount of phosphoric acid excreted during "periods of excitement" in "mental disease." The general statement is made that patients having heavy deposits of phosphates in the urine are "usually subjects of nervous disorders, and frequently of sexual neurasthenia."

The relation of calcium and phosphorus metabolism to "nervous disorders" is uncertain, but it is now known [2] that the amount of calcium or phosphorus excreted in the urine is dependent on very complex physicochemical processes which have no specific relation to the function of the nervous system.

It has long been assumed that the body tissues, in order to function properly, must be bathed by slightly alkaline fluids and that slight variation of the acid-base equilibrium of these fluids is attended by serious disturbances of bodily function. Although the blood remains slightly alkaline, having an average p_H of 7.4, recent observations have shown that cells "can survive and proliferate in an acid medium," and that "the reaction prevailing normally within certain organs of high metabolic activity is frankly acid." [3] From salts in the normal diet are derived basic radicals of sodium, potassium, calcium and magnesium, and certain acidic radicals. The sulphur and phosphorus of proteins are oxidized to sulphuric and phosphoric acids. These radicals are constantly held in such physicochemical combination that the normal

1. Emerson, C. P.: Clinical Diagnosis, ed. 3, Philadelphia, J. B. Lippincott Company, 1911, p. 138. Wood, F. C.: Chemical and Microscopical Diagnosis, ed. 3, New York, D. Appleton & Co., 1911, p. 428.

2. Wilson, D. W.: Neutrality Regulations in the Body, Physiol. Rev. **3**:295 (July) 1923.

3. Rous, Peyton; and Drury, D. R.: Outlying Acidosis, J. A. M. A. **85**:33 (July 4) 1925.

acid-base equilibrium is maintained. Excess of either acidic or basic radical is promptly neutralized and eliminated from the body. Except for volatile acids, practically all acids and bases are excreted by the kidney. In the excretion of acids, phosphorus is one of the predominating acid radicals. It would appear then, that the actual amount of phosphates excreted by the kidney is dependent on the relative amounts of acidic and basic radicals in the body fluids. Thus far no scientific data indicating a specific relationship between the acid-base equilibrium of body fluids and personality disorders have been reported.

So many processes are involved in the maintenance of normal acid-base equilibrium that there may be considerable variation in some of the factors, provided there is compensatory reaction on the part of others. Such variations have been observed both in health and in disease. Some observations of the concentration of calcium and phosphorus in the blood are as follows: injection of insulin produces a decrease of inorganic phosphates [4]; during the later months of pregnancy there is a decrease of calcium and inorganic phosphates, and during lactation an increase of inorganic phosphates [5]; during the period of bone growth and during the period of union of fractures in adults there is a high inorganic phosphate content,[6] and in rachitic children there is a low inorganic phosphate content.[7]

Occasional reference is also made to a relationship of acidosis or alkalosis to mental diseases. Investigation has indicated that there is no demonstrable relation between acidosis and personality disorders.[8] The possibility that conditions of alkali excess or carbon dioxide deficit may have some relation to states of mental tension is suggested by observations of the development of tetany and convulsions in conditions of alkalosis.[9]

In this study blood calcium and phosphorus determinations were made according to the following method.

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4. Wigglesworth, V. B., and others: On the Effect of Insulin on Blood Phosphate, J. Physiol. **57**:447 (Aug.) 1923.

5. Widows, S. T.: Calcium Content of the Blood During Pregnancy, Biochem. J. **17**:30, 1923. De Wesselow, O. L. V.: The Calcium and Inorganic Prosphorus Content of the Maternal Blood During Pregnancy and Lactation, Lancet **2**:227 (July 29) 1922.

6. Tisdall, F. F., and Harris, R. I.: Calcium and Phosphorus Metabolism in Patients with Fractures, J. A. M. A. **79**:884 (Sept. 9) 1922.

7. Howland, J., and Kramer, B.: Calcium and Phosphorus in Serum in Relation to Rickets, Am. J. Dis. Child. **22**:105 (Aug.) 1921.

8. Henry, G. W., and Mangam, E.: Blood in Personality Disorders, Arch. Neurol. & Psychiat. **13**:743 (June) 1925.

9. Koehler, A. E.: Acid-Base Equilibrium, Arch. Int. Med. **31**:590 (April) 1923.

METHOD OF PROCEDURE

All specimens of blood were obtained before breakfast and after at least twelve hours of fasting. The blood for calcium determinations, taken by venipuncture, was received directly into a wide mouthed centrifuge tube. After it had stood for about two hours, the clot was broken up and the specimen was centrifugalized. For phosphorus determinations blood was collected in oxalated tubes. A maximum of 2 mg. of potassium oxalate for each cubic centimeter of blood was used as an anticoagulant.[10]

The calcium content of the blood serum was determined by the Clark and Collip revision [11] of the Kramer-Tisdall method.[12] Serums were usually analyzed the same day and not longer than two days after the specimens were obtained. Calculation: The number of cubic centimeters of 0.01 normal potassium permanganate solution required to produce a minimum of intensity of color in the sample minus the number of cubic centimeters of 0.01 normal potassium permanganate required to produce the same intensity of color in a blank multiplied by 0.2×50 equals the number of milligrams of calcium in 100 cc. of serum sample.

The inorganic, inorganic plus organic hydrolysable and total acid soluble phosphorus were determined by the colorimetric methods of Briggs.[13] As these determinations were made on the whole blood, a one to five dilution of the blood was made, consisting of one part trichloracetic acid, three parts water and one part blood. The filtrates were prepared immediately after collection by filtration on an acid-washed filter paper. Determinations of the inorganic, and the inorganic plus organic hydrolysable phosphorus, were made on the same day the specimens were obtained, and the total acid soluble phosphorus determinations were made several days later. Calculation: (*a*) Inorganic phosphorus: standard set at 25 and containing an equivalent of 0.03 mg. of phosphorus (ammonium molybdate standard).

$$\frac{25 \times 0.03 \times 1 \times 100}{\text{Reading}} = \text{mg. in 100 cc. whole blood.}$$

10. It has been determined that this amount does not interfere with the analysis. Tolstoi, E.: The Inorganic Phosphorus of the Serum and Plasma, J. Biol. Chem. **55**:157 (Feb.) 1923.

11. Clark, E. P., and Collip, J. B.: A Study of the Tisdall Method for the Determination of Blood Serum Calcium with a Suggested Modification, J. Biol. Chem. **63**:461 (March) 1925.

12. Kramer, B., and Tisdall, F. F.: A Simple Technique for the Determination of Calcium and Magnesium in Small Amounts of Serum, J. Biol. Chem. **47**:475 (Aug.) 1921.

13. Briggs, A. P.: Some Applications of the Colorimetric Phosphate Method, J. Biol. Chem. **59**:255 (March) 1924.

(*b*) Inorganic plus organic hydrolysable phosphorus: standard set at 25 containing an equivalent of 0.03 mg. of phosphorus.

$$\frac{25 \times 0.03 \times 2.5 \times 100}{\text{Reading}} = \text{mg. in 100 cc. whole blood.}$$

2.5—since 2 cc. of filtrate used is equivalent to 0.4 cc. of whole blood.

(*c*) Total acid soluble phosphorus: standard set at 20 containing an equivalent of 0.01 mg. of phosphorus.

$$\frac{20 \times 0.1 \times 2.5 \times 100}{\text{Reading}} = \text{mg. in 100 cc. whole blood.}$$

REPORT OF OBSERVATIONS

Calcium determinations were made on specimens from eighty-eight persons, eleven of whom presented no personality disorder. Phosphorus determinations were made on specimens from sixty-eight persons, seven of whom presented no personality disorder. The findings were then tabulated according to the clinical diagnoses at the time the specimens were obtained. No differences between the findings in the two sexes were observed. The results are presented in tables 1 and 2.

TABLE 1.—*Blood Calcium Content in Eighty-Eight Persons*

Clinical Diagnoses	Number of Cases	Mg. per 100 cc. Blood Serum	
		Averages	Extremes
Manic-depressive excitement	16	11.92	9.86-16.55
Manic-depressive depression	12	11.49	9.87-12.50
Involutional melancholia	8	11.11	10.34-12.22
Paranoid condition	1	11.31	.....
Dementia praecox	23	11.47	9.97-13.71
Psychoneuroses	5	11.81	10.76-12.80
Psychoses with psychopathic personality	5	11.78	9.86-12.50
Psychoses with other somatic disease	5	11.39	10.51-12.27
General paralysis	2	11.26	10.98-11.54
Drug addiction	4	11.77	11.29-12.75
Employees	7	10.65	7.89-12.50
Total psychotic group	77	11.56	
Total nonpsychotic group	11	11.05	

TABLE 2.—*Blood Phosphorus Content in Sixty-Eight Persons*

Clinical Diagnoses	Number of Cases	Mg. per 100 cc. of Whole Blood					
		Inorganic		Inorganic Plus Organic Hydrolysable		Total Acid Soluble	
		Average	Extremes	Average	Extremes	Average	Extremes
Manic-depressive excitement	10	3.82	3.02-5.28	10.30	9.19-13.40	23.60	21.27-26.73
Manic-depressive depression	8	3.63	2.40-5.77	9.58	6.38-12.98	21.97	18.51-24.39
Involutional melancholia	5	3.28	2.70-4.08	9.19	7.95-11.06	21.75	20.08-23.69
Paranoid condition	4	4.51	3.88-4.81	9.34	8.33-11.04	22.52	20.41-23.96
Dementia praecox	21	3.69	2.94-5.17	9.54	7.81-11.04	22.39	19.23-26.31
Psychoneuroses	5	3.80	3.51-3.95	9.89	8.52-10.47	23.57	21.84-25.12
Psychoses with psychopathic personality	4	3.25	2.85-3.88	9.64	8.54-10.41	23.57	17.85-27.17
Psychoses with other somatic disease	3	3.08	2.76-3.95	9.38	8.60-10.30	22.01	20.50-23.82
General paralysis	1	3.84	.....	8.93	.....	23.14	.....
Drug addiction	2	4.09	2.83-5.36	9.56	8.72-10.40	23.69	18.65-28.73
Employees	5	4.15	3.54-4.68	9.61	8.15-10.36	22.84	20.33-26.31
Total psychotic group	61	3.68	.....	9.65	.....	22.65	.....
Total nonpsychotic group	7	4.13	.....	9.59	.....	23.08	.....

A study of tables 1 and 2 reveals some interesting variations among the different clinical groups. Of the affective psychoses, manic-depressive excitement has the highest content of blood calcium and phosphorus, while involutional melancholia has the lowest content. This suggests that these variations may be dependent on the quality and quantity of psychomotor activity. These clinical groups were accordingly subdivided.

The dementia praecox group was sufficiently large to permit subdivision according to acuteness or chronicity and according to the commonly recognized clinical types. The results of these subdivisions are presented in table 3.

TABLE 3.—*Calcium and Phosphorus Content of the Blood in Acute and Chronic States*

	Calcium		Phosphorus			
				Mg. per 100 cc. Whole Blood		
Clinical Diagnosis	Number of Cases	Mg. per 100 Cc. Serum	Number of Cases	Inorganic	Inorganic plus Organic Hydrolysable	Total Acid Soluble
Manic	8	12.40	7	3.60	10.47	23.76
Hypomanic	8	11.55	3	4.34	9.92	23.23
Depressed—not agitated	9	11.81	6	3.83	9.99	22.39
Depressed—agitated	3	10.54	2	3.03	8.40	20.78
Involutional melancholia	8	11.11	5	3.28	9.19	21.75
Dementia praecox, acute condition	17	11.46	15	2.67	9.65	22.70
Dementia praecox, chronic deteriorated condition	6	11.48	6	3.56	9.25	21.45
Dementia praecox, paranoid	11	11.50	10	3.91	9.40	22.50
Dementia praecox, hebephrenic	7	11.49	7	3.51	9.29	21.18
Dementia praecox, catatonic	5	11.36	4	3.47	10.31	24.21

COMMENT

When findings so closely approximate the normal average it is difficult to be certain that the variations between the different clinical groups are not accidental. However, the consistency with which these variations were found would seem to merit further consideration. In studying table 3 it is observed that, with the exception of the inorganic phosphates, the calcium and phosphorus content is consistently higher in manic than in hypomanic patients. It is also evident that in the depressed group the agitated patients have a consistently lower calcium and phosphorus content. Since tension and agitation are cardinal symptoms of involutional melancholia, a low calcium and phosphorus content is to be expected in this group.

The relationship of calcium metabolism to motor activity has been amply demonstrated experimentally. Excess of calcium produces tonic

contraction of heart muscle.[14] Deficiency of calcium produces tetany.[15] In the former condition the calcium is supposed to exert a direct stimulating action. In the latter, it is assumed that tetany arises through accumulation of toxic substances ordinarily neutralized by calcium. However, the inter-relationship of elements in the physicochemical processes of the body is so infinitely complex that it is impossible to explain any particular condition on the basis of excess or deficiency of single elements. Nevertheless, there can be no doubt that the quality and quantity of human psychomotor activity has concomitant representation at a physicochemical level.

In view of the relative increase of calcium and phosphorus in conditions in which psychomotor activity is unrestrained (manic), and the relative decrease of these elements in conditions in which increased psychomotor activity represents a protest against overwhelming instinctive and emotional barriers (agitated depression and involutional melancholia), it is interesting to find that the calcium and phosphorus contents in acute and chronic dementia praecox are practically identical and very close to the general average of all groups. It will be observed, however, that in the catatonic group, in which somatic tension is greatest, the calcium content is lowest.

INFLUENCE OF ULTRAVIOLET RADIATION

In recent years the use of ultraviolet radiation, particularly in conditions of calcium and phosphorus deficiency, has greatly increased. The efficacy of this therapy in rickets and allied disorders seems well established.[16] In tuberculous adults the calcium and phosphorus content of the blood is normal and is not affected by ultraviolet therapy.[17] What specific effect ultraviolet radiation has on other diseases remains to be determined. The fact that it does not alter the blood calcium and phosphorus content in personality disorders may have little or no relation to its therapeutic value. Except for those patients necessarily deprived of sunlight, the indications for ultraviolet radiation are dependent on the physical condition of the patient. In such cases the

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14. Howell, W. H.: Textbook of Physiology, ed. 5, Philadelphia, W. B. Saunders Company, 1921, p. 562.

15. Gross, E. G., and Underhill, F. P.: The Metabolism of Inorganic Salts, J. Biol. Chem. **54**:105 (Sept.) 1922.

16. Kramer, B.; Casparis, H., and Howland, J.: Ultraviolet Radiation in Rickets: Effects on Calcium and Inorganic Phosphorus Concentration in Serum, Am. J. Dis. Child. **24**:20 (July) 1922.

17. Howe, M. G., and Medlar, E. M.: The Calcium and Inorganic Phosphorus Content of the Blood Serum in Tuberculosis: Observations on Patients Undergoing Alpine Lamp Treatment, Am. Rev. Tuberc. **10**:408 (Dec.) 1925.

general impression seems to be that this form of therapy is beneficial, and if it is applied properly there are no detrimental effects.

The group of fourteen patients selected for study were those who happened to be receiving ultraviolet therapy during, and in some cases for three months prior to, the time when the specimens were taken. These patients were receiving general radiation from an air-cooled quartz lamp at a distance of 36 inches for a maximum period of twenty minutes every second day. The calcium and phosphorus determinations in this group are presented in table 4.

TABLE 4.—*Calcium and Phosphorus After Ultraviolet Radiation*

Clinical Diagnoses	Calcium, Mg. per 100 Cc. Serum	Phosphorus, Mg. per 100 cc. Whole Blood		
		Inorganic	Inorganic Plus Organic Hydrolysable	Total Acid Soluble
Manic-depressive excitement	11.69	3.80	10.58	23.41
Manic-depressive depression	11.78	3.07	8.83	22.78
Involutional melancholia	11.61	3.51	10.04	22.27
Dementia praecox	10.66	3.60	9.05	23.47
Psychosis with psychopathic personality	12.15	2.98	9.18	23.76
Psychosis with other somatic disease	11.94	3.18	9.38	22.01
General paralysis	11.54	3.84	8.93	23.14
Average with radiation	11.61	3.42	9.37	22.79
Average without radiation	11.55	3.76	9.73	22.62

CONCLUSIONS

1. The calcium and phosphorus content of the blood in personality disorders is within normal limits.

2. In manic states there is a relative increase in the calcium and phosphorus content.

3. In tense, agitated, depressed states there is a relative decrease in the calcium and phosphorus content.

4. The calcium and phosphorus content in both acute and chronic dementia praecox is unchanged.

5. In dementia praecox the calcium content is lowest in the catatonic type.

6. The calcium and phosphorus content of the blood in personality disorders is not affected by ultraviolet radiation.

7. There appears to be a rational basis for calcium and phosphorus therapy in tense, agitated, depressed states.

THE ACTION OF BULBOCAPNINE IN THREE CASES OF PARALYSIS AGITANS AND ONE CASE OF TREMOR OF PARALYSIS AGITANS TYPE

A FURTHER NOTE, WITH A COMPARISON OF THE ACTION OF SCOPOLAMINE, ATROPINE AND PHENOBARBITAL *

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De Jong and Schaltenbrand obtained striking results following the administration of bulbocapnine hydrochloride and phosphate in various types of tremor diseases. As a result of their experiments it seemed desirable to gather further data on the action of bulbocapnine and also to make a comparison of its action with that of other drugs employed in the past for their reputed sedative and hypokinetic action on the central nervous system. In their first paper [1] on the action of bulbocapnine on various types of tremor, these authors give a full account of its experimental history. It has been shown [2] in animals that bulbocapnine causes catalepsy or a cataleptoid state. As a result of these findings, De Jong conceived the idea that these cataleptoid or hypokinetic states might be induced in tremor diseases by the action of bulbocapnine, owing to which the tremor would be diminished or overcome. The formula worked out by Gadamer [3] is reproduced here (fig. 1) from their paper, as well as that of apomorphine, to which it is closely allied.

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* From the laboratory of Prof. Dr. B. Brouwer, Binnen Gasthuis, University of Amsterdam.

1. De Jong, Herman: De Werking van Bulbocapnine op hyperkinetische Toestanden in de Kliniek, Lecture before the Amsterdam Neurologic Association, Nederl. Tijdschr. v. Geneesk **68**:1342 (Sept. 6) 1924. De Jong, Herman; and Schaltenbrand, G.: Die Wirkung des Bulbocapnins auf Paralysis Agitans und andere Tremorkranke, Klin. Wchnschr. **3**:2045 (Nov. 4) 1924.

2. Frohlich, A., and Meyer, H. H.: Ueber Dauerverkürzung der gestreiften Warmblutermuskeln, Arch. f. exper. Path. u. Pharmakol. **87**:173, 1920. De Jong, Herman: Ueber Bulbocapninkatalepsie, Klin. Wchnschr. **1**:684 (April 1) 1922; Over Katalepsie en Bulbocapnine-werking, Nederl. Tijdschr. v. Geneesk. **2**:794 (Aug. 25) 1923.

3. Gadamer, J.: Ueber Corydalisalkaloide, Arch. d. Pharmazie **240**:14, 1902.

METHOD

The method used here—an ordinary muscle pelotte attached to the recording muscle by a wide leather band—is the same as that previously used by De Jong.[4] The thenar muscle was always used. The initial position of the pelotte on the muscle was marked with ink, as a change of the position of the pelotte on the muscle might well alter the amplitude of the tremor. Therefore, whenever the pelotte was even slightly disarranged during the course of an experiment, the data obtained were rejected.

A tube with a fitting piece connected the pelotte to a Marey's tambour. The lever of the tambour was so arranged as barely to touch the surface of the smoked drum. In this way friction was minimized. While this method is less absolute in accuracy than electromyographic studies, De Jong and Schaltenbrand [5] found it quite adequate for deter-

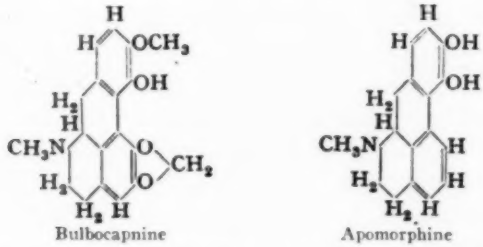

Fig. 1.—The formula of bulbocapnine, as worked out by Professor Gadamer of Marburg, and the formula of apomorphine to which it is closely allied, reproduced from the paper of De Jong and Schaltenbrand. "It differs from it (apomorphine) in its ortho sidechains. It is moreover dextrorotary, whereas apomorphine is levorotary."

mining the rougher differences in the tremor before and after injection. A smooth line, while not indicating the complete absence of tremor, showed clearly the effect of the drug, and was always in accord with clinical observations. In their cases, as well as in our own, the tremor had at times to be elicited by forced movements of the arm, such as a swinging of the arm which held the pelotte. The apparatus employed was quite undisturbed by these movements. Consequently, consistent results were obtainable, as the same movements could be performed to elicit the tremor both before and after injection.

4. De Jong, Herman; and Schaltenbrand, G.: The Action of Bulbocapnine on Paralysis Agitans and Other Tremor Diseases, Neurotherapie (Supplement of "Psychiat. en neurol. Bladen") **28**:61, 1924.

5. De Jong, Herman; and Schaltenbrand, G.: Further Clinical Investigation with Bulbocapnine, Neurotherapie 1925; Weitere Versuche mit Bulbocapnine in der Nervenklinik, Deutsche Ztschr. f. Nervenh. **86**:129, 1925.

DOSAGE

Schaltenbrand,[6] in experiments on himself, found that 350 mg. could be taken without undue toxic effect. After this dosage he felt moderate fatigue and a slowing of psychic function. In the series of cases reported from Amsterdam, 200 mg. at times produced the symptoms reported above. Consequently, 200 mg. of bulbocapnine hydrochloride or phosphate was regarded as the maximum dose. In our series, in refractory cases in which no toxic symptoms could be observed, the dosage was pushed up to 300 mg. At times a moderate degree of drowsiness lasting from two to three hours followed these larger doses, but no other toxic effects were noticed. One may find, therefore, even in persons suffering from the same disease, marked variations both in responsiveness and in tolerance to bulbocapnine.

PREVIOUS RESULTS

It has long been known that scopolamine decreases both the tremor and the rigidity in paralysis agitans. Cobb[7] injected $\frac{1}{250}$ grain (0.26 mg.) of scopolamine hydrobromide intravenously in cases of paralysis agitans and recorded his results with electromyograph. De Jong and Schaltenbrand next compared reactions to bulbocapnine and to scopolamine in seven cases[5] and reported certain striking differences: 1. Bulbocapnine acts, if at all, in from ten to fifteen minutes after injection; while scopolamine always began to act approximately one-half hour after injection. 2. In four cases, scopolamine was more effective than bulbocapnine. In one case, bulbocapnine was quite without effect. In one case, no difference in the effect of the two drugs could be demonstrated. In two cases, bulbocapnine was more effective than scopolamine, and in one case scopolamine was quite without effect. 3. In general they found that scopolamine acted with more intensity than bulbocapnine, and that the duration of the effect was longer.

They emphasize that their reports on scopolamine are based on single experiments. They conclude that in prolonged therapy bulbocapnine has probably the advantage of a constant effect, the absence of habit formation and the absence of the need of ever-increasing doses. They observed also no unfavorable symptoms on its withdrawal. Scopolamine therapy, as is already known, has all of the foregoing drawbacks.

On the other hand Gordon Holmes[8] has observed after intravenous scopolamine therapy a diminution of rigidity lasting some hours. This

6. De Jong and Schaltenbrand (footnotes 1 and 4).

7. Cobb, Stanley: Electromyographic Studies of Paralysis Agitans, Arch. Neurol. & Psychiat. **8**:247 (Sept.) 1922.

8. Oral communication to the authors.

amelioration has never been observed after bulbocapnine therapy.[9] As a result of the work outlined above, the following comparative studies were made of four suitable cases.

PROTOCOLS OF THE FOUR PATIENTS STUDIED

CASE 1.—Th., a man, aged 65, with a history of paralysis agitans of one and one-half years' duration, had a marked tremor in the right hand, and also in the

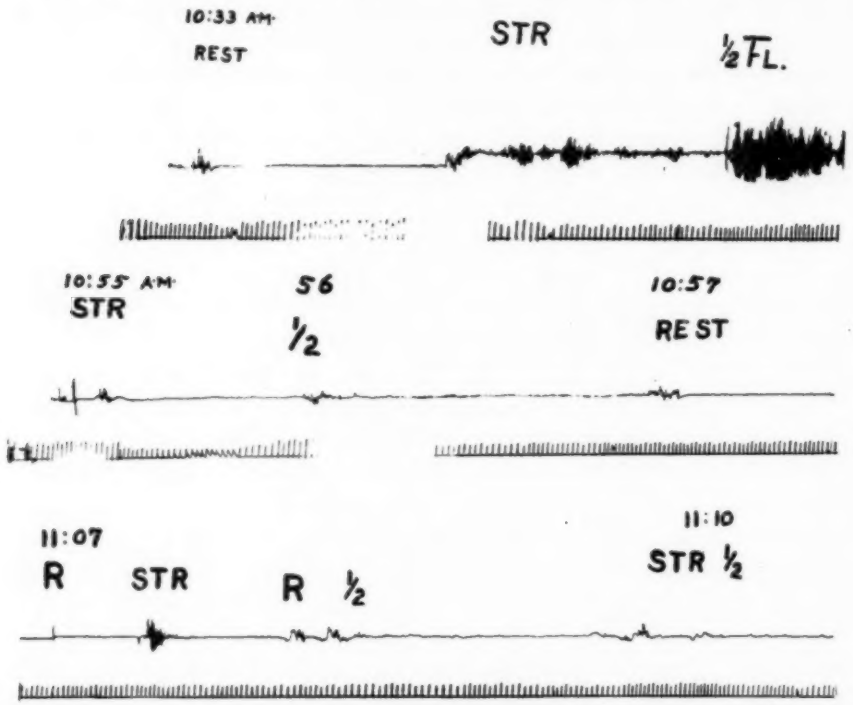

Fig. 2.—Three registrations on patient Th. The first was taken before the administration of bulbocapnine, 150 mg.; the second eight minutes after injection, and the third twenty minutes after injection. The effect of the drug on the tremor is clear. *Rest* and *R* indicate that the arm is held at the side and resting on the knee, with the patient sitting down; *STR* means that the arm is extended vertically over the head; *½ FL* means that the arm is abducted from the body and horizontal and that the forearm is held in half flexion. These three symbols indicating the position of the arm during the experiment are used in all the following figures.

left hand after exertion or emotion, with generalized rigidity. The patient moved "en bloc."

This patient had been treated by De Jong and Schaltenbrand, and the tremor had disappeared after the administration of from 150 to 200 mg. of bulbocapnine

9. Other references of interest concerning the effects of bulbocapnine are: Peters, F.: Untersuchungen über Corydalisalkaloide, Arch. f. exper. Path. u. Pharmakol. **51**:130, 1904. Schaltenbrand, G.: Ueber die Bewegungsstörungen bei akuter Bulbocapninvergiftung, ibid. **103**:1, 1924.

hydrochloride. The same dosage was again tried and our results were consistent with their findings. The tremor curve flattened out within from eight to twelve minutes after the injection of the drug, and the effect lasted from one to two hours (fig. 2). This experiment was repeated several times and the same results were always obtained.

Scopolamine was next tried, in a dose of 0.25 mg. The tremor was diminished within thirty minutes after injection. The tremor line was flat within an hour after injection, and remained so for four and one-half hours. It gradually reappeared, but even six hours after injection it had not reached its full height (fig. 3). This experiment was repeated with similar results which lasted from three to four hours.

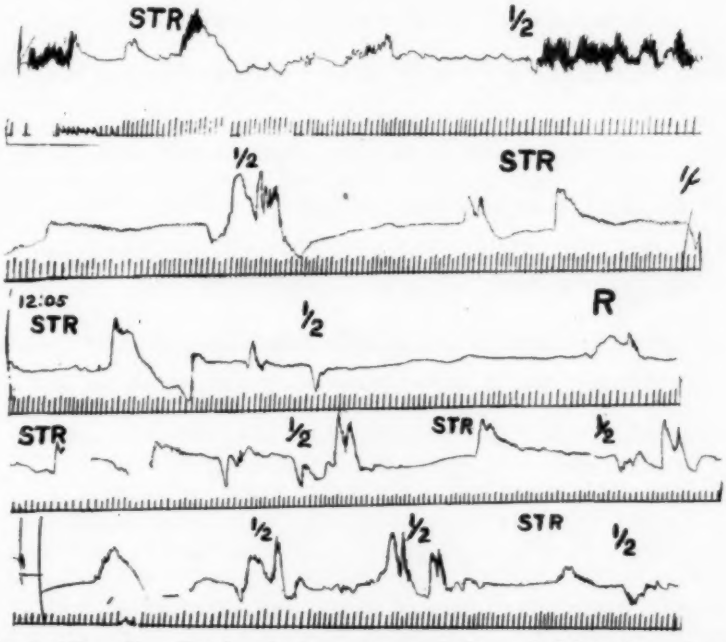

Fig. 3 (Case 1).—Tracing 1 shows tremor before administration of scopolamine; tracing 2 shows disappearance of tremor one-half hour after injection of scopolamine, 0.25 mg.; tracing 3 shows absence of tremor one hour after injection; tracing 4 shows absence of tremor three hours after injection; tracing 5 shows tremor barely appearing four and one-half hours after injection. The tremor only reappeared in considerable degree six hours after injection and was still lower than before injection.

Atropine was then administered, 1.0 mg. being given in two doses of 0.5 mg. thirty minutes apart. Although there was an increase in the pulse rate and a dryness of the mouth, there was no effect whatever on the tremor.

Phenobarbital in doses of 75 mg. had no effect, as indicated in the accompanying table.

Morphine, 15 mg., had no effect.

Case 2.—Alk., a man, aged 62, gave a typical history of paralysis agitans of about four years' duration combined with a severe trigeminal neuralgia. When the neuralgia was severe the tremor was more marked. The tremor was not

constant, occurring every three to five minutes at rest and always after fatigue, as for example when the arm was outstretched or half flexed.

Bulbocapnine was administered. The first doses of from 150 to 200 mg. did not stop the tremor, but increased the interval between the attacks of tremor during rest to from five to eight minutes. In one instance, after 300 mg. was given, the patient was free of rest tremor for forty minutes. The free interval during rest treatment was conditioned by the degree of pain due to the trigeminal neuralgia from which the patient was suffering. During this interval the tremor was elicited as usual when the arm was outstretched or flexed. In this patient bulbocapnine did not stop the tremor; it had a slight effect, however, in that the interval at which the tremor appeared, was lengthened (fig. 4).

Scopolamine was administered in doses of 0.25 mg. No effect on the tremor was noticed, though the patient was very drowsy and had difficulty in keeping awake.

Atropine, 0.5 mg., given in two doses over intervals of one-half hour, had no effect on the tremor.

Phenobarbital had no appreciable effect. One-half hour after administration the tremor height was somewhat diminished, but one hour after injection the tremor line was higher than before its administration. Both of these records were, however, within the limits of normal variation.

Fig. 4.—Tracing of the typical rest tremor of the patient Alk. one hour after a total of 300 mg. of bulbocapnine had been injected in two doses. Five minutes after the second injection the rest tremor ceased and did not reappear for forty minutes, at which point it resumed its normal amplitude.

Case 3.—H. C. Van R., a man, aged 70, who had suffered from paralysis agitans for eighteen years, on examination showed a typical pill-rolling tremor and generalized rigidity. The patient could walk only with assistance, and there was marked propulsion and retropulsion, as well as drooling and excessive sweating.

Three administrations of bulbocapnine were made. In one there was a very slight reduction of the tremor line fifteen minutes after 300 mg. of the drug had been given. This reduction, however, was not sufficient to be regarded as a definite effect of the injection. Two other records, after 225 mg., showed not the slightest lowering of the tremor line.

Scopolamine, on the other hand, showed a definite effect (fig. 5). The tremor was clearly decreased in volume, and the effect lasted two hours and forty-five minutes after injection. Three and one-half hours after injection the tremor was at full height. A second injection with scopolamine showed similar results.

Atropine, 0.5 mg., was given in two doses of 0.25 mg. each at an interval of one-half hour. The patient ordinarily suffered from excessive salivation and drooling, but after the second dose complained of a dry mouth. The tremor curve showed not the slightest effect of the drug, as indicated in the accompanying table.

Phenobarbital, 75 mg., had no effect on the tremor.

Case 4.—Sch., a man, aged 61, with no apparent rigidity and no suggestion of a parkinsonian facies, had a tremor at rest which was increased by the arm being outstretched or placed in half flexion. This tremor had appeared more than a year before.

Bulbocapnine showed a definite effect within fifteen minutes after the injection of 150 mg. Nothing more than pulse oscillations could be seen on the record (fig. 6). A repetition of this experiment gave identical results.

Atropine, 0.25 mg., had no effect on the tremor. In a second injection, 0.5 mg was given, and negative results were again obtained.

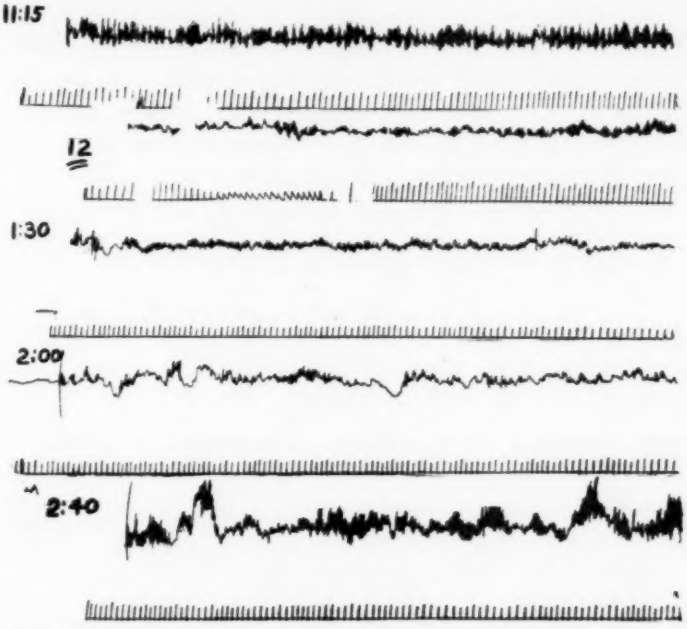

Fig. 5 (Case 3).—Tracing 1, at 11:15, is a control curve; tracing 2, at 12 noon, shows decrease of tremor forty-five minutes after injection of scopolamine, 0.25 mg.; tracing 3 shows continued decrease two hours and fifteen minutes after injection; tracing 4 shows continued result two hours and forty-five minutes after injection; tracing 5 shows return of tremor to preinjection height, three hours and thirty-five minutes after injection.

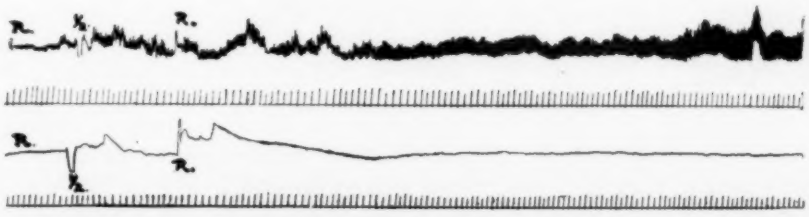

Fig. 6 (Case 4).—The first tracing was made before injection of bulbocapnine; the second tracing, twenty minutes after injection of 150 mg. of bulbocapnine, showed a complete flattening of the tremor line.

Phenobarbital, 75 mg., showed a probably negative effect. The tremor line showed lower oscillations, but not lower than the lowest oscillations in the control tracings. A repetition with phenobarbital, 100 mg., showed a totally negative result.

Scopolamine, 0.25 mg., showed not the slighest effect.

SUMMARY AND CONCLUSIONS

Four patients, of whom three showed the typical parkinsonian syndrome, and the fourth a tremor of the parkinsonian type but no other signs, were treated with a group of drugs known to have a quieting action on the central nervous system. Of these four patients, two showed a striking amelioration of the tremor after bulbocapnine and two showed a questionable improvement, as indicated in the accompanying table.

Two patients showed a striking improvement after scopolamine, and two were entirely refractory to scopolamine.

All four were refractory to atropine and phenobarbital.

Comparison of the Effects Produced on the Tremor by the Drugs Used

	Bulbocapnine	Scopolamine	Atropine	Phenobarbital	Morphine
Case 1	+	+	—	—	—
Case 2	±	—	—	—	..
Case 3	±	+	—	—	..
Case 4	+	—	—	—	..

+ indicates flattening of the tremor line; ±, moderate lowering of the tremor line; —, no appreciable change in the tremor line.

Morphine was administered only to one patient, who had responded particularly well to bulbocapnine and scopolamine. It was found to have no effect on the tremors. This drug was not employed further in this study because, as is only too well known, even in the most careful hands it is apt to be more disastrous than beneficial in chronic cases.

Judging from the four patients examined, bulbocapnine and scopolamine alone can be regarded as of real therapeutic value. Atropine and phenobarbital are of no value.

It is evident that cases refractory to both bulbocapnine and scopolamine are not rare. The cause of this is not known. In the refractory cases, twice the dose given under ordinary conditions was administered, and though the patients were conspicuously drowsy, the tremor was unaffected.

LOSS OF WEIGHT: ITS IMPORTANCE AS AN EARLY SYMPTOM IN GENERAL PARALYSIS*

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In the course of obtaining detailed clinical histories of the onset and precommitment evolution of the disease in a series of patients suffering from general paralysis of the insane, I have been impressed by the relative frequency with which loss of weight was mentioned spontaneously as having had a place, and sometimes a prominent one, among the early symptoms presented. It has seemed worth while to look further into its occurrence in view of the fact that loss of weight has received no very special consideration, so far as I am aware, in discussions of the symptomatology of early general paralysis.[1] Furthermore, loss of weight, as a symptom, possesses in itself considerable significance in that it indicates some fundamental even if ill-defined disturbance of metabolism;[2] the occurrence, as a more or less prominent manifestation in a due proportion of cases, of general paralysis should serve to direct the emphasis to a more general biologic approach to the study of the raison d'être of that disease. "Metasyphilis" does not affect the central nervous system only, nor is general paralysis solely an organic disease of the brain, but rather it represents an involvement—obscure in its nature, it is true—of the entire organism. Finally, loss of weight is noteworthy in being a manifestation which characterizes conspicuously the onset and early course of relatively few diseases. Hence, I have investigated further the incidence of loss of weight in a series of cases of general paralysis, the amount of loss and its relationship in point of time to other symptoms. For this purpose I have utilized the clinical histories of seventy-four male patients with general paralysis which have already been reported in another connection.[3]

Thirty-eight of these seventy-four patients suffered from loss of weight at some time subsequent to the earliest signs of the onset of the

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* From the New York State Psychiatric Institute, Ward's Island, N. Y.

1. Albrecht, O.: Diagnose der beginnenden progressiven Paralyse, Wien. med. Wchnschr. **73**:1757, 1813 and 1950, 1923. Claude, H.; Targowla, R., and Santenoise, D.: La phase pré-symptomatique de la paralysie générale, Presse med. **32**:81 (Jan. 30) 1924. Menninger, K. A., and Menninger, W. C.: Symptom Analysis in Paretic Neurosyphilis: A Study of One Hundred and Sixty-Six Comparable Consecutive Cases, Am. J. Syph. **9**:104 (Jan.) 1925.

2. Compare, in a slightly different connection, Kraepelin, E.: Das Rätsel der Paralyse, Naturwissenschaften **12**:1124 and 1125 (Dec. 12) 1924.

3. Bunker, H. A., Jr.: Incipient General Paralysis: A Study of the Earliest Symptoms Presented by Seventy-Four Cases, Am. J. M. Sc. **171**:386 (March) 1926.

disease and prior to their commitment. In twenty-two of these, loss of weight constituted essentially the earliest departure from the normal observed by the nearest relative (usually the wife of the patient). In the remaining sixteen, the onset of loss of weight either could not be definitely dated in relation to other symptoms, possibly in many instances owing to the gradual character of the loss, or was known to have occurred only later in the evolution of the disease.

Apart from any question of the amount of weight loss, the simple fact is arresting that in 50 per cent of these patients a loss of weight sufficient to be noticeable to others took place during the precommitment period of the disease; in 30 per cent of the seventy-four cases the decline in weight was essentially the earliest symptom to appear (in 17.5 per cent the very earliest). Indeed, the foregoing understates the case for three reasons: in many instances direct questions regarding loss of weight were purposely avoided; there were instances in which the informant, even when interrogated specifically on the point, was ignorant of the fact that loss of weight had occurred, although there was sometimes evidence of another kind, later to be discussed, that this had actually been so; cases have not here been included in which a loss of weight not attested by the informant might have been deducible from a comparison of the patient's weight on admission to the hospital with his alleged customary weight in health, since the possible errors involved in such a procedure are obvious. Even patients whose customary weight was subsequently ascertainable beyond a reasonable doubt and in whom a loss of weight was accordingly demonstrable with certainty from the discrepancy between this weight and the admission weight (these additional patients were at least five in number) have not here been included, since it was the qualitative aspect of the matter—the fact that in some early cases of general paralysis there occurred a loss of weight sufficient to attract the notice of the intimates of the patients—rather than the question of the actual proportion of cases in which early loss of weight took place, which engaged our interest primarily. The diagnostic criterion has, in a word, rested in this: whether the informant was or was not able to say that the patient had appeared to her observation to have lost weight.

As to the twenty-two patients in whom loss of weight was either the earliest departure from the normal state of health to be observed (thirteen cases [8]) or was the second earliest symptom to appear (nine cases [8]). In three, no estimate of the actual amount lost was possible; in the remaining nineteen cases, some idea was gained of the amount of weight that had been lost. It is permissible to suppose that a loss of 5 pounds (2.3 Kg.) would in general be the smallest which could attract attention, while in many instances 10 pounds (4.5 Kg.), perhaps the smallest loss to have any real significance, would more nearly repre-

sent this minimum. In only two of these nineteen patients did the loss of weight appear to have amounted to as little as 10 pounds; in both these cases, moreover, this amount is a conservative estimate based on data rather more meager than in almost any of the remaining cases. Among the latter (seventeen in number) the loss of weight ranged from 10 to 15 pounds (4.5 to 7 Kg.) in six cases, to from 25 to 45 pounds (11.3 to 20.4 Kg.) in four cases—representing something like from 7 to 28 per cent of the customary weight of the patient in health.

Brief notes of a few of the more striking examples may be given.

REPORT OF CASES

CASE 1.—D. M. seemed to his wife, toward the end of December, 1922, to have lost considerable weight; this was some three weeks after an attack of herpes zoster, but about two months before the appearance of definite mental symptoms, and practically a year before the patient's commitment, Dec. 6, 1923. His weight was found to be only 160 pounds (72.6 Kg.), whereas ordinarily it was 189 pounds (85.7 Kg.), clothed. Four months later his weight had fallen to 147 pounds (66.7 Kg.); in September, 1923, it was 139 pounds (63 Kg.); on Dec. 17, 1923, it was 131 pounds (59.4 Kg.), stripped. We have here, then, a loss of nearly 50 pounds (22.7 Kg.) within the space of some fourteen months, which represented about 28 per cent of the patient's normal weight.

CASE 2.—E. A. M., in January, 1923, weighed 190 pounds (86 Kg.), and it seemed to his wife that even then he was beginning to lose weight; but how much above this his ordinary weight was she could not state with certainty. Three months passed before any other symptom developed, nine months before frank mental symptoms made an appearance, and ten months before commitment. By March, 1923, his weight had fallen to 163 pounds (74 Kg.), so that "people did not know him on the street." Nov. 16, 1923, his weight was 160 pounds (72.6 Kg.), stripped. He had lost 30 pounds (13.6 Kg.), most of it within three months, the total representing about 16 per cent of his normal weight.

CASE 3.—A. M. W. had a sudden attack of aphasia (with residual speech defect) on Nov. 11, 1918. Although his weight prior to 1910 was more than 195 pounds (88.5 Kg.), for some years he had weighed from 165 to 170 pounds (75 to 77 Kg.), and was known to have been of this weight in 1918. Toward the end of 1919, however, he was found to weigh only 150 pounds (68 Kg.). By April, 1924, he had lost an additional 15 pounds (7 Kg.), and by July 1 another 5 pounds (2.3 Kg.). Definite mental symptoms had first appeared some time in February, 1924; by August it was said his weight had fallen to 115 pounds (52 Kg.). On October 13, however, he weighed 127 pounds (57.6 Kg.), stripped. He had accordingly lost not less than 30 pounds (13.6 Kg.) in all, or about 19 per cent of his normal weight; but this process, almost consistently progressive, had covered an interval of four and one-half years.

CASE 4.—The wife of H. R. was positive that loss of weight was the earliest change noticed by her in the patient, and it dated back to the autumn of 1922 His ordinary weight was about 185 or 190 pounds (84 or 86 Kg.). After January, 1923, he seemed to lose weight more rapidly, so that "his clothes just hung on him" and "everybody in the neighborhood noticed it." Although he complained of indigestion and headaches in January, 1923, he presented no definite mental symptoms until April, 1923; commitment was postponed until November,

1924. On Nov. 21, 1924, he weighed 149 pounds (67.6 Kg.), stripped. Here, then, was a loss of not less than 35 pounds (16 Kg.), or 19 per cent of his normal weight, in about two years and two months.

Case 5.—E. G. was known to have weighed about 145 pounds (66 Kg.) in 1918. In the light of subsequent events his wife realizes that he began to lose weight, although very gradually, as early as the autumn of 1921; this was nearly two years before any further symptoms appeared, more than three years before frank mental symptoms developed and almost four years prior to commitment. In the summer of 1923, however, at about the time that other symptoms began to become evident, he commenced to lose weight much more rapidly; "before we knew it he was down to 130 pounds." Two years later, on July 16, 1925, he weighed 116 pounds (52.6 Kg.), stripped. This represents a total loss of weight of not less than 20 pounds (9 Kg.), about 14 per cent of his weight in health, extended over a considerable period of time. That this weight of 116 pounds did not arise from the fact that the patient was by that time moribund is demonstrated by the circumstance that the patient achieved an excellent remission under treatment and has recently been discharged from the hosiptal.

These five cases might be regarded as exceptional; but they at any rate show how prominent a feature of oncoming general paralysis marked and early loss of weight may sometimes constitute, even though this be the fact in perhaps only a small proportion of cases.

It is clear that loss of weight may take place in a very gradual manner at the outset and proceed over a considerable period of time before becoming sufficient to attract the notice of intimates of the patient; after such an interval the loss of weight may then, with the advent of some other symptom or symptoms, go forward somewhat more rapidly. When this is the course of events, it may be only good fortune—as, for example, in cases 3 and 5—that makes it possible to date the true onset of the decline in weight and thus the chronologic relationship of this onset to the appearance of other symptoms. Hence, if loss of weight is specifically stated by the informant as having been the first symptom observed in the patient, the statement is one which ipso facto can hardly be doubted; but when this is not the case, it does not necessarily follow that the loss of weight described had not been going on for a considerably longer time than the informant was aware.

With regard to the sixteen additional cases with a history of loss of weight not occurring as the first or second symptom to be noted, the foregoing reservation probably holds true in some; but in others it seems rather definite that loss of weight did not begin to take place until after the appearance of more or less outspoken mental symptoms.

Case 6.—E. N. exhibited his first symptom, in the form of irritability, in May or June, 1924. His usual weight was in the neighborhood of 150 pounds (68 Kg.), and as late as September, at least three months after the onset of mental symptoms, he was known to have weighed 153 pounds (69.4 Kg.). But by November 3 his weight had fallen to 125 pounds (56.7 Kg.), stripped; so that he had lost about 20 pounds (9 Kg.), or some 14 per cent of his normal weight. in the space of two months.

Much as in the first group of cases, the amount of weight lost by the patients of this second group ranges from an average of from 8 to 12 pounds (3.6 to 5.4 Kg.) in five cases, to 45 pounds (20.4 Kg.) in one case, representing from 5 to 28 per cent of the patients' customary weight.

There remains to be mentioned the much increased appetite, or bulimia, which characterized the precommitment evolution of the disease in a number of these patients—a symptom which certainly has the advantage, compared with loss of weight, that it almost instantly attracts the notice of the informant and obviously makes a marked impression. Indeed, the symptom is only the more convincing in the light of the rather interesting fact that with only three exceptions it occurred in men who were normally rather heavy eaters. As is natural, the bulimia varied considerably in degree. Perhaps the slightest case was that of a man who, about the time that frank mental symptoms developed and nearly two years after the initial appearance of certain other symptoms, acquired the habit of stopping for a lunch on his way home from work at night; he would then eat a more than usually large dinner, while there were certain times in particular when he "couldn't seem to get enough to eat." This patient, whose usual weight was about 180 pounds (81.6 Kg.), was barely 10 pounds (4.5 Kg.) underweight on admission to the hospital. Possibly the most extreme instance was that of a patient who developed a ravenous appetite about four months after the onset of loss of weight, at a time when he had already lost some 30 pounds (13.6 Kg.). About seven months later, when he had lost nearly 50 pounds (22.7 Kg.), his appetite still further increased, so that, in fact, he became something of a curiosity to the neighbors. At meals he took two and sometimes three large helpings of every dish, would sometimes eat 2½ pounds (1 Kg.) of steak at a sitting, ate between meals as well, would consume eight or ten apples during the evening and would awake after a few hours' sleep and complain of hunger. Like Samuel Johnson, who "swallowed his tea in oceans," this patient at the same time acquired the habit, previously foreign to him, of taking from fifteen to twenty-five cups of tea in twenty-four hours. Several other patients showed also a special predilection for fruit of various kinds; one patient manifested his bulimia principally in his consumption of meat, and one patient exhibited an intense craving only for candy and sweets, of which he was normally very fond.

Of the thirty-eight patients described above, there was in twelve a definite history of bulimia. Five of these belonged in the group of twenty-two patients whose loss of weight constituted either the first or the second departure from their usual state of health; the remaining seven are found among the sixteen patients in whom loss of weight seems to have been a symptom of rather later development. Although

the numbers involved are too few to warrant any conclusion, it is possible that the rather more protracted period over which loss of weight took place in those patients in whom it was a very early symptom was responsible, through the consequent very gradual character of this loss, for the failure to evoke in many of them a compensatory (?) increase of appetite of abnormal degree.

The difficulty of assigning a precise date to the onset of loss of weight in the majority of cases interferes with an estimate of the relationship in point of time between the loss of weight and the bulimia. It is possible in some instances that the latter made its appearance before loss of weight had proceeded to any material extent; on the other hand, it has been definitely ascertainable in a few cases that pathologically increased appetite did not manifest itself until the patient's weight had been declining for many months. It might be interesting, indeed, to know the minimum amount of weight loss capable of calling out this possibly compensatory mechanism; but this is not possible from the data here available.

In this connection must be mentioned seven cases not previously dealt with, in which a definite history of bulimia was obtainable without, however, any loss of weight having occurred, to the informant's knowledge. In several of the patients there was, nevertheless, presumptive evidence of some loss of weight, amounting in each instance to possibly as much as from 10 to 12 pounds (4.5 to 5.4 Kg.). Whether, then, in these seven cases loss of weight occurred but escaped the notice of the informant, or whether the loss was so fully counterbalanced by the increased food intake as to be essentially nonexistent or so slight as to be imperceptible to others, cannot be said; probably either may be true on occasion.

Case 7.—E. M. developed a psychosis with great suddenness after a blow on the head on Aug. 23, 1924; it was reasonably clear that no other abnormality preceded this outbreak save for a single suspicious episode some three weeks before. During the five days subsequent to the head trauma he manifested a much increased appetite and "could eat a whole steak at once." On admission, September 26, he weighed 169 pounds (76.7 Kg.). There was no means of knowing whether this was less than his usual weight. Feb. 20, 1925, he was discharged, and immediately resumed his former occupation. On August 23 he weighed 178 pounds (80.7 Kg.), and on November 8, 185 pounds (84 Kg.). It is therefore probable that at the time of his commitment he had lost at least 8 or 9 pounds (3.6 or 4 Kg.), possibly as much as 15 pounds (7 Kg.).

We have, then, a total of nineteen patients out of the seventy-four (25 per cent) with a history of bulimia, with or without a degree of preceding weight loss appreciable to the informant.

Too many factors, obviously enough, enter the question of remissions in treated cases of general paralysis to make a favorable outcome of this

sort reducible to a single principle. Nevertheless, it is perhaps of some interest, although possibly a mere coincidence, that of the seven patients just mentioned in whom it may be said that increased food intake seemed to have more or less counterbalanced loss of weight, five have responded very satisfactorily to treatment, one has done moderately well and only one, with a very advanced case at the time of admission, has failed to improve. When the twelve patients with a history of both bulimia and loss of weight are viewed from this standpoint, it is found that six have done well and six have not. But, on the other hand, of the six who did well, four were patients in whom there was, in spite of a definite history of loss of weight, little objective evidence at the time of admission of such loss; so that in these four patients, likewise, increased food intake had seemingly acted in a more or less successfully compensatory manner. Of the six patients in this group who did not respond to treatment, four exhibited a marked loss of weight in spite of bulimia. It is of course undeniable that a number of patients who had no history of bulimia and who had suffered a considerable loss of weight achieved nevertheless very satisfactory remissions following treatment; there are five such patients in our series to date, even though it remains true that the majority of our remissions have been recruited from among those who presented no history of loss of weight or whose loss in weight was distinctly moderate. It is not intended, however, to lay undue stress on this point; it is mentioned merely as a tentative suggestion that the behavior of the body weight in cases of early general paralysis may play a certain part in the response of the patient to subsequent treatment: while marked loss of weight during the evolution of the disease is not always or necessarily of poor omen for a satisfactory response to active therapy, a favorable influence in this regard may possibly inhere in the fact that little or no loss of weight has taken place or that the loss has been to a considerable extent offset by the increased food intake incident to bulimia.

SUMMARY

1. In a series of seventy-four male patients with general paralysis, loss of weight, often well marked, but often also very gradual, was essentially the earliest abnormality noted in twenty-two cases. In seventeen of these cases the loss of weight ranged from an average of from 10 to 15 pounds (4.5 to 7 Kg.) in six cases to from 25 to 45 pounds (11.3 to 20.4 Kg.) in four cases, representing some 7 to 28 per cent of the ordinary weight of the patient in health, the modal value being 14 per cent.

2. In sixteen additional cases, loss of weight had definitely taken place but was a later manifestation.

3. In twelve of these thirty-eight cases there was also a history of greatly increased appetite or bulimia, which usually developed some time after the onset of loss of weight, and which was in a few cases successful in counterbalancing to a considerable extent the preceding weight loss.

4. In seven additional cases, in which no known loss of weight had taken place, there was likewise a definite history of bulimia.

5. There is a possibility of a relationship of these findings to the therapeutic response of the patient.

Clinical Notes

PARALYSIS OF THE OCULOPUPILLARY SYMPATHETIC FIBERS AND THE OPHTHALMIC DIVISION OF THE TRIGEMINAL NERVE, PRECEDED BY OPHTHALMIC HERPES ZOSTER

Report of a Case *

CHARLES S. POTTS, M.D., PHILADELPHIA

In 1924, J. G. Raeder[1] of Oslo reported five cases of paralysis of the oculopupillary sympathetic fibers associated with lesions involving the trigeminal nerve, to which he gave the name "paratrigeminal" paralysis. To explain this occurrence he gives the anatomic relations as follows: "The cervical trunk takes origin in the ciliospinal center between the sixth cervical and fourth dorsal segments of the cord. The fibres run from here through the rami communicantes to the upper dorsal ganglion, and through the ansa Vieussenii and the cervical trunk to the upper cervical ganglion. Some of the fibres accompany the external carotid artery to the skin of the face, and oculopupillary fibers reach the base of the brain with the internal carotids, as the plexus caroticus internus. Here some of these fibers run as fine filaments to the gasserian ganglion and join the first branch of the trigeminal nerve; others accompany the oculomotor nerve. Those that join the trigeminal nerve pass through the naso-ciliary and long ciliary nerves to the eye and innervate the dilator pupillae, while the filaments which join the oculomotor nerves supply the involuntary superior and inferior palpebral muscles. On their way through the carotid canal, some fibres probably pass by way of the deep petrosal nerve to the sphenopalatine ganglion and innervate the orbital muscle."

The symptoms in Raeder's first case, which came to necropsy, were pain in the left eye, left temple and left side of the head, and paralysis of the left ocular sympathetic and of the left motor branch of the fifth nerve. There were also diplopia and vomiting. The lesion was a tumor in the left middle fossa, involving laterally the gasserian ganglion. In front it extended approximately to the superior orbital fissure, and backward to the posterior limit of the middle fossa. The third, fourth and sixth nerves were displaced, and the growth covered the internal carotid artery. In addition to the gasserian ganglion, the nerve filaments connecting the carotid plexus with the trigeminal and oculomotor nerves were injured. The lesion in the other four cases, the symptoms of which were similar but not identical, there being sensory impairment, was assumed to be in the same location. They did not come to necropsy. The point is made that in these cases only ocular sympathetic fibers were damaged. This is explained by the assumption that the sympathetic fibers were injured after the branches to the other organs of the face had already parted from them.

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* Read, and patient shown, at the meeting of the Philadelphia Neurological Society, Jan. 22, 1926.

1. Raeder, J. G.: "Paratrigeminal" Paralysis of Oculopupillary Sympathetic, Brain **47**:149 (May) 1924.

REPORT OF CASE

A man, aged 59, who was an iron worker by occupation and had always been in good health until about a year before coming under observation, was referred to me Jan. 12, 1924, by Dr. A. C. Sautter, an ophthalmologist of Philadelphia. At that time he had been ill for three days with what was thought to be influenza. This was associated with severe, constant pain in the right frontotemporal region and eye. The skin was hypersensitive. On the third day a vesicular eruption appeared on the forehead, and in a few days scabs formed. During that night the right eye suddenly felt "as if it popped out and then went back again." He then noticed that this eye looked peculiar.

Since the illness there had been a disagreeable itching sensation on the right side of the forehead. Examination showed the right palpebral fissure markedly narrowed and the eyeball retracted. The pupil was contracted and did not dilate when shaded nor from the use of cocaine; it did contract when stimulated by light. No change in sweating on the two sides of the face had been noticed. Over the right forehead were a number of depressed, whitish scars which at first, he stated, were dark in color. There was marked diminution of pain and tactile sense in this region and the cornea was anesthetic. Temperature sense was not tested. With the exception of some pyorrhea, nothing else of importance was found. The points of special interest to me are the occurrence of the unusual combination of symptoms described by Raeder, and the still more unusual occurrence of herpes zoster as a probable cause.

There was evidently a paralysis of the oculopupillary sympathetic fibers and weakness of the function of the ophthalmic division of the trigeminal nerve. The case, therefore, presents Raeder's criteria for what he terms "paratrigeminal" paralysis. The lesion was an inflammation with succeeding degeneration, probably due to the specific infection that causes herpes zoster.

COMMENT

In case 19 of Head and Campbell's series,[2] the distribution of the eruption was similar to that in this patient. They found evidences of inflammation and degeneration in part of the gasserian ganglion on the affected side, and some evidence of degeneration in the sensory root. The symptoms of case 4 of Raeder's series were attributed by him to inflammation.

The occurrence of paralysis of the sympathetic in connection with herpes zoster must be very unusual. In Bing's "Gehirn und Auge," also in his textbook on nervous diseases, is shown a picture of a patient with frontal herpes and paralysis of the oculopupillary fibers. He does not comment on the case; Raeder refers to it in his paper. Jelliffe and White[3] state, "Herpes corneae, herpes ophthalmicus, keratitis neuroparalytica, are among the eye affections due to implication of vegetative fibers, located chiefly in the trigeminus sheaths or in the gasserian ganglion." I found no references to this combination in a search through most of the current textbooks on neurology and dermatology. Radcliffe Crocker mentions complete ophthalmoplegia, transient hemiplegia and paralysis of the deltoid and of the hand as occurring with herpes zoster and paralysis of the third and seventh nerves in cases with no other evidence of organic disease of the nervous system, but says nothing of sympathetic paralysis.

2. Head and Campbell: The Pathology of Herpes Zoster, Brain **23**:351, 1900.

3. Jelliffe and White: Diseases of the Nervous System, ed. 4, Philadelphia, Lee & Febiger, 1923, p. 164.

While the symptoms in this case, exclusive of the sympathetic paralysis, are typical of herpes zoster, the question arises: Could the infection be that of epidemic encephalitis? The constitutional symptoms in this disease often resemble those of mild influenza. Herpes is said to occur, and Cadwalader[4] has reported two cases of encephalitis in which there was bilateral sympathetic paralysis. In his cases the lesions were not peripheral as in mine. Personally, I incline to the view that the infection in my case was that which usually causes herpes zoster whatever that may be. It seems evident that paralysis of the oculopupillary sympathetic fibers may occur from lesions in the gasserian ganglion or its vicinity and, therefore, may have localizing value.

The patient was seen again (Jan. 20, 1926), and there was no change in the symptoms.

THE UNCINATE SYNDROME*

WILLIAM G. SPILLER, M.D., PHILADELPHIA

Since the work of Hughlings Jackson we have recognized the symptoms of irritation of the uncinate gyrus, and in 1904, when Jackson's uncinate group of fits had received little attention in this country, I[1] wrote on the subject and reported several cases. Jackson said in his description: "In cases of this group there is at the onset of the paroxysms a crude sensation of smell or one of taste, or there are movements of chewing, smacking of the lips, etc. (sometimes there is spitting). In some cases of this group there is a warning by what is known as the epigastric sensation, a crude development of a systemic sensation; this warning sometimes occurs along with a crude sensation of smell or with the chewing, etc., movements. Different varieties of this group of cases depend, I suppose, on discharge lesions of different parts of what I call the uncinate region."

The paralytic symptoms of a lesion of the uncinate region are less well known. They are impairment of smell and taste on the side of the lesion; homonymous lateral hemianopia in the half fields opposite the lesion, from involvement of the optic tract on the side of the lesion; probably signs of pituitary disease; some gradually progressive paralysis of the side of the body opposite the lesion, from implication of pyramidal tract fibers; possibly oculomotor paralysis or signs of cerebellar disorder. If the tumor implicates the chiasm, the cutting of the visual fields may assume some other form than homonymous lateral hemianopia.

The following case is an example of this symptom-complex.

REPORT OF CASE

J. W. P., aged 22, referred to me by Dr. Charles J. Craythorn and Dr. Horace Bellis of Trenton, Dec. 9, 1925, had noticed in February, 1925, some weakness of the right hand, which gradually increased. In May he noticed

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4. Cadwalader, W. B.: Occurrence of Bilateral Sympathetic Ophthalmoplegia, Its Significance in Lethargic Encephalitis, J. A. M. A. **74**:1315 (May 8) 1920.

* Case presented before the Philadelphia Neurological Society, Dec. 16, 1925.

1. Spiller, W. G.: Am. Med. **7**:474-475 (March 19) 1904.

some weakness of the right lower limb and right side of the face. He said he had had diplopia about two years previously lasting about one week. Since the onset of the paralysis, he has had on an average of one headache a week lasting about twenty-four hours.

On my examination great impairment of smell was found in the left nostril for oil of wintergreen, vinegar and valerian; he recognized these substances slightly, but was unable to name them. His recognition was prompt with the right nostril. No abnormal condition was detected by a nasal examination. There was no evidence of sphenoid or ethmoid disease. Taste for sugar and vinegar was greatly impaired, and salt was not recognized at all on the left side of the tongue; but on the right side all these substances were recognized and named promptly.

There were marked pituitary disturbances. The features were coarse and suggested acromegaly, and the upper teeth showed unusual spacing. The hands and feet were very large, the hips were rounded and of the feminine type and the pubic hair in its distribution was of the same type. The man had no hair on his chest and little in the axillae; he needed to shave only twice weekly. The genitalia were normally developed. He had a tendency to obesity. There was no impairment of objective sensation. The man had some asynergia and was unable to rise from a supine to a sitting position with his upper limbs across his chest, but he did not have cerebellar catalepsy. The tendon reflexes were all greatly diminished or lost. Dr. Fewell found right homonymous lateral hemianopia; vision in the right eye was 6/12, in the left eye 6/6; the disks were well defined.

The right upper and lower limbs and right side of the face were distinctly weak; the dynamometer registered in the right hand 40, and in the left 130. The basal metabolic rate was — 14.4 per cent.

The roentgen-ray report from Dr. Pancoast was: enormous enlargement of the pituitary fossa, presumably due to pituitary encroachment on the sphenoid sinuses, with disappearance of the dorsum sellae and posterior clinoid processes. Measurements of the pituitary fossa could not be made.

The diagnosis made by me was: large pituitary tumor implicating the left optic tract and not the chiasm, pressing on or invading the left uncinate gyrus or cerebral peduncle so as to impair the pyramidal tract fibers on their passage from the internal capsule to the foot of the left cerebral peduncle, but not extending far enough posteriorly to involve the oculomotor nerve. The tumor must greatly impair the left uncinate gyrus, causing pronounced impairment of taste and smell on the left side. The diminution of the tendon reflexes and the asynergia were regarded as secondary cerebellar signs, possibly caused by hydrocephalus or by pressure on fibers connected with the anterior or middle cerebellar peduncle.

A transfrontal operation was performed later by Dr. Frazier; a large pituitary tumor and cyst were found and the latter was evacuated.

A CASE OF UNUSUAL BRACHIAL PLEXUS PALSY

PHILIP LEWIN, M.D., AND HARRY ARKIN, M.D., CHICAGO

M. K., white, born Feb. 19, 1924, was the first child of healthy parents and a full term baby. Delivery was a face presentation and the membranes ruptured thirty-six hours before. Forceps and strong traction were required to effect a delivery, the mother being under ether anesthesia for two and a half hours. The abnormal position of the upper extremities was noticed immediately after delivery. The mother first noticed it on the fifth day when the baby held the

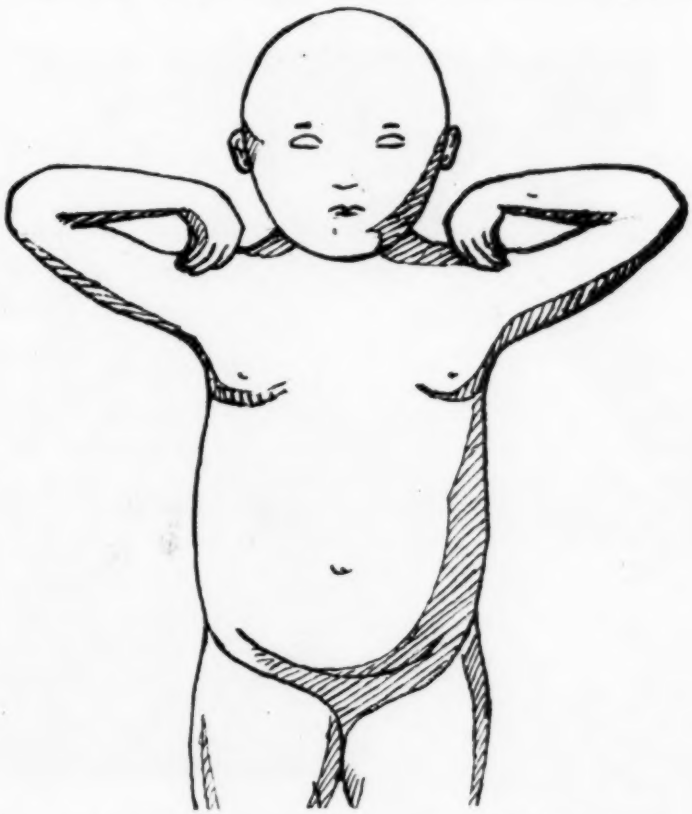


Fig. 1.—Patient before treatment was instituted.

arms abducted, the elbows flexed, the wrists dropped and deviated ulnarward, and all fingers flexed. The bases of the thumbs touched the acromion processes. This position was maintained continuously except during sleep when the hands were under the chin.

Examination.—On April 28 the child, then nearly ten weeks old, was seen by Dr. Lewis J. Pollock, who reported normal findings except for the arms, as follows:

"The arms are abducted and make an angle of 120 degrees with the thorax. They are rotated outward, the forearms flexed and pronated. The hands are

flexed at the wrist; the thumbs are abducted, the fingers flexed. There is a contracture of the palmaris longus on the right, less pronounced on the left. Extension of the fingers and wrist is difficult on both sides. When the arm is passively adducted it abducts suddenly and forcefully when released, and becomes externally rotated. Passive adduction of the arm is associated with movement of the scapula throughout the entire movement. There is absence of movement in the extensors of the wrist, fingers and thumbs. There is no obvious sensory change. To the galvanic current the extensors of the left wrist react with moderate rapidity, but despite the position of abduction of the arm the deltoid muscle does not show any obvious contraction to the faradic current. The left deltoid does not react normally to galvanism" (figs. 1 and 2).

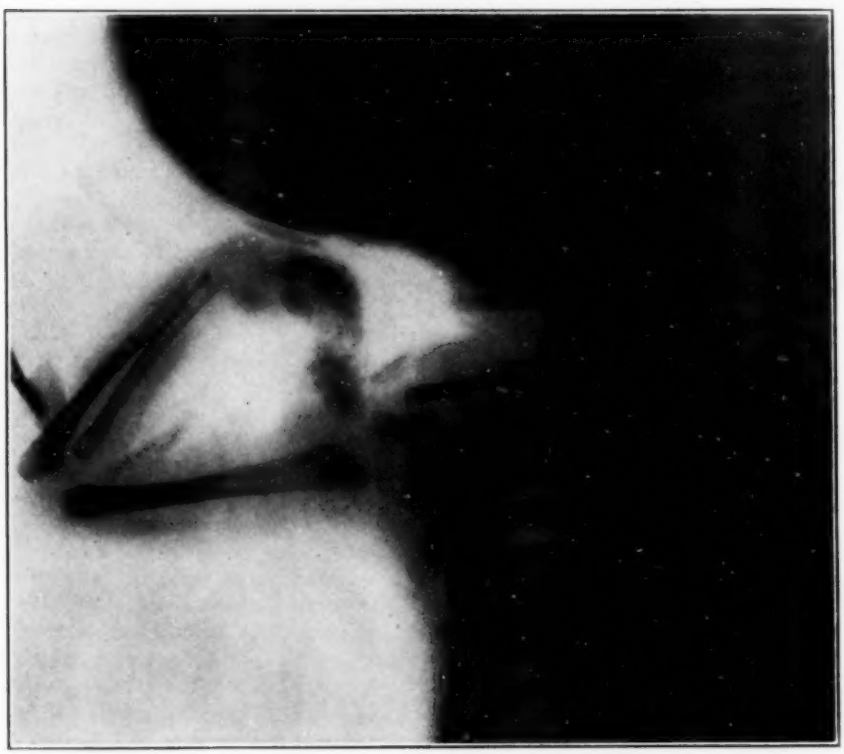


Fig. 2.—Normal shoulder joint and characteristic attitude of upper extremity.

Examination by us on May 6, 1924, revealed nothing else worthy of note. There was no dislocation of either shoulder.

Treatment.—Without anesthesia the arms were very gently completely adducted, the elbows extended and the entire extremities held against the sides by a swathe of cloth. Measurements were made for cock-up splints of aluminum to overcome the wrist drop, to extend the fingers and to abduct the thumbs. Within twenty-four hours the arms remained in adduction and did not again return to the abducted position when released, as they formerly did. The splints were kept on for thirteen months, being removed only for massage and electric treatments. These were begun July 22. The slow sinusoidal cur-

rent was applied three times a week to the extensors of the wrists and fingers; after five months, only twice a week until the present time (January, 1926). Massage by a competent nurse was given three times a week to the shoulder girdle and the triceps and extensors of the wrists and fingers. In addition, massage was given by the mother twice a day.

Progress.—At the age of 8 months the child had almost normal use of its upper extremities including the hands. It reached in all directions for objects and was able to hold light objects. There was marked improvement in the response to electric stimulation. Some shortening (contracture) of the flexors of the fingers remains at present.

A similar though less marked case was reported by Jolly.[1] In this instance a face presentation was changed by manipulation to an occiput posterior. There was no deflection of the back or manipulation of the arms. Soon after birth, abduction of both arms and paralysis of both forearms and hands were noticed and thought at first to be due to luxation of the shoulder joints. Jolly concluded, however, that the case was one of unusual palsy and not a dislocation. He believed the paralysis was due to injury of the seventh root, caused by trauma during a face presentation, with extreme lordosis, which latter was increased by the so-called Thorn grip. He stated that a high grade lordosis might affect the nerve roots at their emergence through the convexity between the sixth and seventh cervical vertebrae. The patient did not make good recovery of function.

Summary and Conclusions.—The case, therefore, is one of bilateral seventh cervical root birth palsy. The pathologic condition is due to trauma to the nerve roots during a difficult delivery. The injury must be in the roots of the plexus, and with a symmetrical lesion it must be close to the points of exit of the roots from the spinal column, possibly within the spinal canal. It is of interest that in both Jolly's and our cases there was a face presentation.

1. Jolly: Charité Ann. **21**, 1896.

Obituary

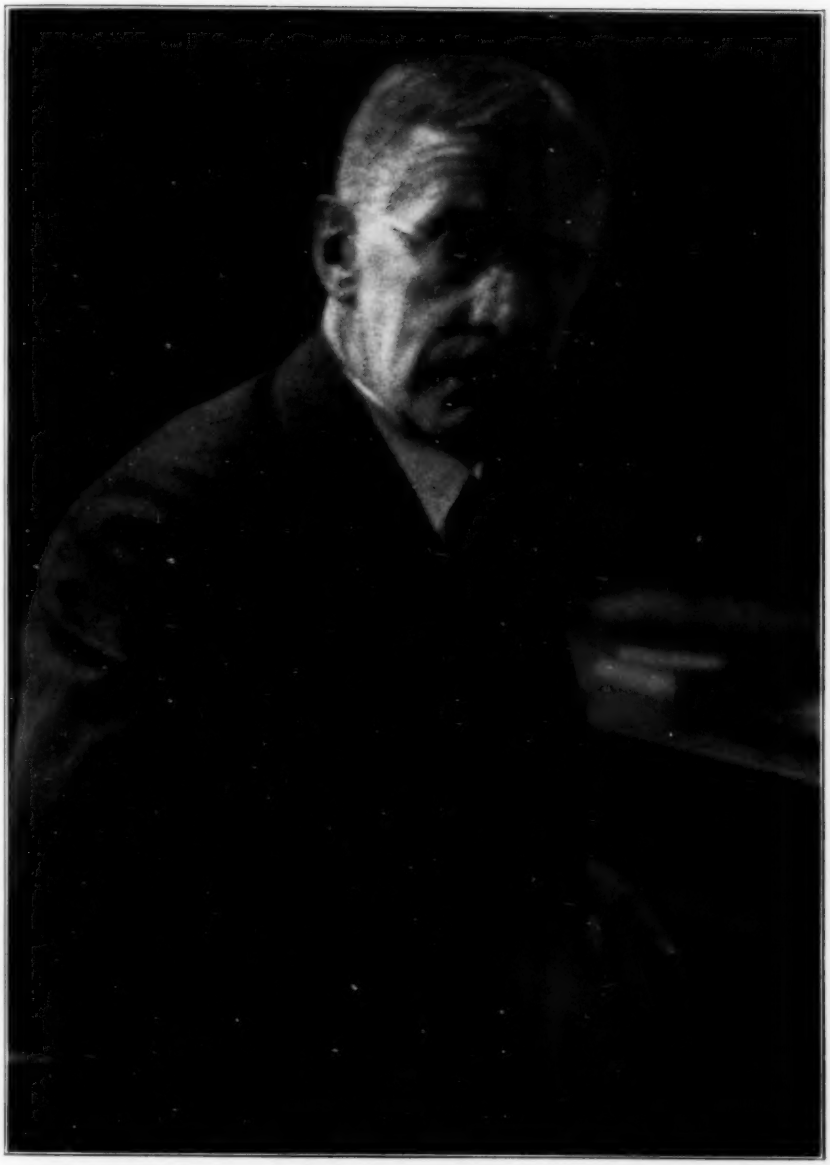
HENRY M. THOMAS, M.D.

1861—1925

Henry M. Thomas of Baltimore died on June 21, 1925, at Blue Ridge Summit, Pa., shortly after entrance on his sixty-fifth year. He is survived by his widow (Josephine Gibson Carey Thomas) and by their two sons, Dr. Henry M. Thomas, Jr., (Associate in Clinical Medicine in the Johns Hopkins University) and E. Trudeau Thomas (a Rhodes Scholar at Oxford, now Master at St. George's School, Newport, R. I.).

The death of Dr. Thomas, clinical professor of neurology in the Johns Hopkins University Medical Department and visiting neurologist to the Johns Hopkins Hospital for the last thirty years, has made, in the neurologic departments of those institutions, a void that is keenly felt by all who knew him. Whatever the developments in clinical neurology in the future at Johns Hopkins may be, there will always be reason for pride and satisfaction in the fact that the first occupant of the clinical chair in that subject had the first-rate diagnostic ability, the teaching clarity, objectivity, and impressiveness, and the qualities of mind and heart that characterized the life and work of Henry M. Thomas. Sometime, Johns Hopkins may have an elaborate neurologic institute, wards especially provided for organic neurologic patients and an extensive equipment and a liberal endowment for neurologic teaching and research—all of which were dreamed of and longed for by Thomas; but never can she acquire for her staff a more painstakingly accurate and adequate examiner of patients, a more conscientious and beloved instructor of students, a wiser guide in therapy or a more helpful consultant with colleagues than the man who for the three decades just past taught and practiced neurology with a very limited equipment and budget in the outpatient department of her hospital. Men like Henry M. Thomas are all too rare; and it is fitting that those of us who have had the privilege of knowing such men should, on occasion, express our admiration and appreciation of them.

Born in Baltimore, May 25, 1861, the son of Dr. James Carey Thomas (a medical practitioner and a trustee of the Johns Hopkins University) and Mary Whitall Thomas, Henry M. Thomas was derived from sturdy Quaker ancestry, recognizable in the parental names by those familiar with the "Society of Friends" in America. A distin-



HENRY M. THOMAS

guished sister, M. Carey Thomas, was for many years the president of Bryn Mawr College. Educated first in private schools in Baltimore, and later in Haverford College, Thomas received the A. B. degree from the Johns Hopkins University in 1882, where he was a fellow student with Councilman and Howell and where he studied biology under Martin, chemistry under Remsen and Shakespeare under Lanier. He obtained his M. D. degree from the University of Maryland School of Medicine in 1885. In the latter school, under the teaching of Professor Miles, his interest in neurology was aroused and on graduation he went to Europe to work in neurology in Heidelberg and in Vienna. It was fortunate that during this year of postgraduate study he came under the especial influence of a great master of neurology, Prof. Wilhelm Erb of Heidelberg. Even a superficial knowledge of the qualities of Erb as investigator, teacher and practitioner should suffice to reveal his influence on this young student who was later to be clinical professor of neurology at Johns Hopkins University.

On returning to Baltimore he joined a group of men that surrounded, and were inspired by, Professor Welch in the Pathological Institute of the Johns Hopkins University, a group that at that time included Councilman, Halsted and Mall. With them he worked during his spare time, of which he had much, for private patients were but few in number and his appointment as attending physician to the insane at Bay View Hospital required only occasional visits.

When William Osler took up his duties at the Johns Hopkins Hospital in 1889, he was looked on as the guiding star of all the younger clinicians. Thomas' association with the great internist soon ripened into warm friendship, and after the Johns Hopkins University Medical Department was opened, Osler gave him charge of the neurologic teaching. Speaking of those early days Thomas himself has said: "The coming of Osler ushered in the complete realization of long deferred hopes. . . . What good there is in me as a teacher and a physician I owe to him."

Though he could scarcely be called a prolific writer, Thomas made, on the average, one publication each year. His clinical papers reveal clearly his passion for accuracy of observation as well as his critical acumen. They deal almost wholly with organic diseases of the nervous system (inflammatory, toxic-degenerative, neoplastic and vascular). In his case reports he picks out the novel and unique features and lays emphasis on them and thus saves them from obscure burial in medical literature. And no feature was ever reported as new or rare before he had made an exhaustive review of the publications of others for identical or similar instances. Of his contributions may be mentioned especially the papers on Raynaud's disease with convulsions and hemoglobinuria (1890); on cerebrospinal syphilis with unusual lesions in the spinal

cord (1891); on congenital facial diplegia (1898); on obstetrical paralysis (1900); on chorea with retinal embolism (1901); on neurofibromatosis with paralysis and muscular atrophy of the arms and legs (1903); on thrombosis of the posterior inferior cerebellar artery (1908); on lead poisonings (1904, 1914), and on multiple neuritis (1898, 1907). His careful statistical study (1899) of the cases of tabes studied at the hospital in which he worked was noteworthy as was also his report on a case of tetany in pregnancy in which he suggested that the occurrence of tetany during pregnancy may depend on "an insufficiency, absolute or relative, in the action of the thyroid gland or like structures," thus exhibiting both his caution and his prescience. His capacity for adequate historical surveys of medical and biographic topics was illustrated by his papers on the anatomic basis of the Argyll Robertson pupil (1903); the decussation of the pyramids (1910); the life of Elizabeth Fry (1919), and the address (as President of the American Neurological Association) on Sir Charles Bell (1911). But if he had been asked which of his publications he, himself, valued most highly, he would have said the article on Diseases of the Cerebral Blood Vessels, published in Osler's Modern Medicine in 1910. In this he was able to bring together for the use of students and practitioners the results of his large personal experience and of his careful studies of medical bibliography bearing on the vascular disorders of the brain. This paper will always stand as adequately representative of the state of medical knowledge on this important subject at the time that it was written.

As a teacher, Thomas led his assistants and his pupils gradually and systematically to acquire the methods of accumulating data (anamnestic and physical) regarding single patients; to learn how to utilize the data thus acquired for localizing lesions within the nervous system and for determining the nature and pathogenesis of the disease processes that give rise to them, and, based on secure diagnosis, to plan the modes of therapy most likely to be helpful. Those whom he instructed not only respected him for his knowledge and skill but, attracted by his strong and lovable personality, also admired him greatly as a man, and developed a genuine affection for him. Many of those who were favored by invitations to his home will never forget the charming hospitality extended to them both by the teacher and by his wise, understanding and gracious wife. There are not a few who gratefully acknowledge that their ideals of life and of work owe much to the example and influence of the Thomas household, observed and felt during their formative period.

As a practitioner, Thomas' work was restricted largely to neurologic consultations, though he did undertake the personal supervision of the therapy of a certain number of patients suffering from either organic or

functional nervous disorders. Though his training had been predominantly concerned with organic nervous diseases, his interest in psychoneurotic and in mildly psychotic patients was keen and he understood very well how by common sense methods to help them. He welcomed, however, the gift to the Johns Hopkins University of the Phipps Psychiatric Institute and the advent as professor of psychiatry of Adolf Meyer, whose training in the anatomy and pathology of the nervous system as well as in psychiatry ensured, he believed, protection from the vagaries and mystic trends that tended to characterize some of the modern workers in psychology and psychiatry.

Handicapped as he was by a severe migraine and by a slowly progressive pulmonary tuberculosis (which, as early as his twenty-seventh year, had necessitated a sojourn at Saranac and which culminated in the pulmonary fibrosis and terminal infection that caused his death), he knew the discomforts, the limitations and the thwarting of ambitions for which chronic invalidism can be responsible. But instead of reacting with bitterness, he showed that his sympathy for the sufferings of others had only been increased by his own experiences and he maintained a steady cheerfulness, indeed a sunny optimism, not preached but exhibited, which was most wholesome in its effects on his patients. In this particular, he resembled closely his intimate friend, Dr. E. L. Trudeau (who also suffered from severe migraine and from tuberculosis), whom he had learned to know, to admire and to love, when taking his cure in 1888. Those who have had opportunity to observe both Trudeau and Thomas and who have read the address of the former on "Optimism in Medicine" scarcely can have failed to recognize the similarities in sincerity, in courage, in sympathy and in cheerfulness of the two men. One can only regret that these noble souls were so disadvantaged as they were by bodily ills; though this regret may be mitigated, perhaps, by a sense of gratitude for the inspiring example they set of fortitude in bearing those ills and of turning personal misfortune into good for others.

LEWELLYS F. BARKER, Baltimore.

Abstracts from Current Literature

MIGRAINE AND EPILEPSY. EMIL REDLICH, Wien. klin. Wchnschr. **39**:21-25 (Jan.) 1926.

The close relationship between migraine and epilepsy has been referred to since the time of Charcot, Féré, Möbius and others. Among the more recent writers on the subject are L. R. Müller, Greving and Richter. The reason for assuming a relationship of the two diseases is that frequently the family history of a patient with migraine shows epilepsy, and, also, the same person often suffers from both maladies.

A patient who has had migraine since childhood may have epileptic seizures later in life. This is especially true of persons whose migraine attacks are of an ophthalmic nature (according to Féré and Flatau), and in whom the attack begins with a definite scotoma. With the onset of epilepsy the migraine may diminish or entirely disappear. Then with the cessation of the epileptic attacks the migraine may recur. Likewise, a patient who has suffered from epilepsy may have the attacks cease and migraine take their place. This has been referred to by Strohmayer as "mitigation of epilepsy."

Cases in which both forms appear under the same guise or are directly combined are of special importance. The attacks set in with a typical scotoma and merge into an epileptic seizure. In such cases the migraine has been looked on as a symptom of the epilepsy and referred to as an epileptic migraine. Frequently also an epileptic attack is followed by a typical migraine headache.

Both diseases respond to the same therapy. Bromide has long since been recognized as effective in migraine and phenobarbital, which has been used in epilepsy, has more recently been recommended for migraine. The author uses a mixture of bromide and phenobarbital effectively.

Many authorities deny a relationship between migraine and epilepsy. F. Schultze asserts that migraine and epilepsy are not identical. He claims that since there is no unconsciousness and no Babinski phenomenon in migraine, even in migraine of long standing, there cannot be the anatomic change in the brain which is responsible for the symptoms of epilepsy.

If F. Schultze, Christiansen and others consider the frequent occurrence of migraine and epilepsy in the same family or in the same person as a mere coincidence, it must be admitted that it occurs all too frequently and does not explain a combination of the two forms in the same attack. Further, Christiansen is not justified in considering a trauma of etiologic moment in such cases. Schultze and Velter claim there is a difference between the scotoma of migraine and the visual aura of epilepsy as optic hallucinations always enter into the latter picture.

It is difficult to say which etiologic and pathologic points are to be considered in establishing a relationship between epilepsy and migraine. When the family history includes both affections this is simple enough. However, such findings occur in only a part of the cases. In the remaining cases it is not sufficient to assume that the patients belong to a neuropathic family, and it is therefore necessary to seek the connecting link in the pathogenesis of both diseases. If we are thoroughly honest we must admit that we are still very much in the dark in this matter.

Gowers was under the impression that the severe migraine headache uncoupled an epileptic seizure. Flatau rightly denied this because in other

diseases, even in the face of equally severe headaches, epileptic attacks do not occur. Further, an epileptic attack is not always ushered in by a headache; and also both forms most frequently occur independently of one another.

The author's investigations have led him to believe that there is a pathologic reaction in the brain and that typical mechanisms are set free by certain irritations. He also believes that there are indications for direct localization. The causative irritation, however, is still unclear and may be looked for in the metabolism, endocrine influences and similar functions.

In both migraine and epilepsy certain manifestations are observed which are brought about by irritation of certain brain areas. From this standpoint it is possible that the same or a similar irritation causes a reaction in or about the same brain area, and therefore it is understandable that both forms of the disease might appear in the same person or that a transference of the irritation may cause a combination of the two attacks.

L. R. Müller and Greving claim that all symptoms of both forms may be localized in the neighborhood of the midbrain, of the root ganglia and of the third ventricle. They assume increased pressure in the third ventricle as causing the attack.

It is certain that various forms of aura of the epileptic attack, the unconsciousness, the clonic twitching and the many other symptoms, as the psychic forerunners and after-effects of epilepsy, point to the cortex. That the cortex and subcortical centers enter the scene was suggested as far back as Binswanger. It is difficult to ascribe to the subcortical centers the headaches, but even more difficult to explain the scotoma and associated symptoms of most migraine attacks, such as aphasia, hemiparalysis and hemianesthesia, on this basis.

Richter, and more recently Hahn and Stein, have endeavored to explain the migraine attack as being brought about by a change in sympathetic tonus in the lower cervical ganglion and of the cerebral vessels, especially the vertebral artery. This artery, by way of the posterior cerebral artery, supplies the occipital lobe, and here the migraine symptoms manifest themselves, as also in the pons and medulla oblongata and a part of the midbrain, and from these points the other symptoms of migraine are explainable.

There have been many objections to this theory and the author does not believe that the relationship of migraine and epilepsy is adequately explained on such a basis.

Taking into consideration the sympathetic nature of migraine and epilepsy, it would be simpler to assume that they are brought about by an organic brain process. This is generally accepted for epilepsy, but it is also known that migraine attacks occur with organic diseases of the brain. This is to be considered in those cases without heredity which appear later in life. Migraine occurs with brain tumor, cerebral arteriosclerosis and especially cerebral syphilis, as also with hereditary syphilis, metasyphilis, tabes and progressive paralysis. (It is a question if the author is not stretching a point here.) As epileptic seizures also occur in connection with these processes, a combination of the two in the same person is not difficult to explain.

The author cites in great detail a case of a patient, aged 23, who had epileptic attacks and migraine coming on in 1923. There was no previous history of epilepsy or migraine, but her father died of tabes. She was given eight neo-arsphenamine injections and treated with bromides and phenobarbital, and made a good recovery. This patient evidently suffered from ophthalmic migraine associated with psychic epilepsy.

The literature on migraine, especially ophthalmic migraine, includes a long list of symptoms indicating a cerebral irritation from which homonymous hemianopia, aphasia, hemiplegia, hemiparesthesia and hemianesthesia may develop. As a rule these symptoms are transitory. However, cases have been reported in which a bilateral homonymous hemianopia continued after a migraine attack. Von Valkenburg reported a case of a girl, aged 24, who had suffered from epilepsy since her thirteenth year in whom, after an attack, there remained a left-sided hemianopia, which gradually confined itself to a hemianopia of the lower quadrant. (Such residuals are not uncommon.)

One is rightly inclined to attribute such an end-result to a vessel spasm. However, Wilbrand-Sanger suggests that such a condition brings up the question of a symptomatic migraine, especially vessel changes as in arteriosclerosis and syphilitic endarteritis. The author believes that there is a possibility of some such condition in the case which he has described, in that the patient's father had syphilis, although syphilitic findings, except for the gold curve, were negative in the patient's case.

As consistent with the author's case, Forster has expressed himself as believing that an irritative focus causes the migraine and also the epilepsy. To set off the attack there must be some special irritation. This may be different for the one or the other of the attacks so that with the introduction of one or the other stimulant some one attack occurs, then the other; and they may likewise combine. Many of the influences which bring about or cause migraine also cause epilepsy. These include nutritional errors, disturbances of metabolism, exogenous poisons, as alcohol, etc. In the female the generative apparatus plays a part in the production of migraine. It is well known that migraine and also epilepsy, frequently appear at the time of puberty. Both diseases frequently appear with the menses and cease at the climacteric. This is especially true of migraine.

The pathology and pathogenesis of migraine and epilepsy are by no means exhausted. Kammerer has recently pointed out what a difficult subject is being dealt with in migraine, and the subject of epilepsy is even more difficult. Even a case such as the author's, which seems relatively simple to explain, does not give a complete insight into the subject, and we must patiently wait for further study before arriving at any conclusion as to the relationship between the two; but it may be that the direction indicated here is the correct one.

MOERSCH, Rochester, Minn.

STUDIES ON HAIR, WITH SPECIAL REFERENCE TO HYPERTRICHOSIS. C. H. DANFORTH, Arch. Dermat. & Syph. **12**:195 (Aug.) 1925.

In this study, the fifth of a series on the growth of the hair, the author discusses at length what is known on the subject. He finds that little information is available regarding the factors that control or influence hair production, and as little regarding the simpler phases of the problem—the normal duration of life of each hair, or the periods of activity or quiescence in the individual follicle. In studying these factors it would seem necessary to settle the particulars of the cycle of normal hair production. The duration of this cycle has been estimated on the basis of the rate of growth of individual hairs, the frequency with which hairs fall out, and on the cyclic activity of the follicles. Studies on the rate of growth of hair, after cutting or shaving, show that the rate is fairly constant for a given region and size of hair, and it is easy to estimate the time necessary for a hair to attain its maximum length. This would be the life-span of the hair, were it not for periods of quiescence. That

quiescent periods do occur is verified by histologic appearances, but their duration is unknown. It is known that terminal hairs on the ears and eyebrows occasionally show a quiescent period of three months, a considerably longer time than the hair takes to attain its maximum length. These observations have not been extensive enough to establish such a time period as a general rule.

It has been assumed generally that external factors, such as shaving, have a stimulating effect on the rate of growth and on the production of hair, but the large majority of controlled studies show no evidence for the truth of this assumption; in fact, activity and quiescence of the follicle appear to proceed at a regular rate, regardless of the length or condition of the external hair.

A similar negative result follows other external factors, such as the application of cosmetic creams and the effects of heat, cold or sunlight, though in the last case some slight stimulating action may result from the simultaneous hyperemia of the skin. In regard to the artificial application of actinic rays, the roentgen rays appear to have a slight direct stimulating action. The results said to be obtained with violet radiation are very questionable and those obtained with ultraviolet rays are probably the result of the concomitant hyperemia. In fact the consideration of the available data leads to the conclusion that external stimuli, except roentgen-ray therapy, whether applied to the hair itself or to the skin, have little or no direct effect on hair growth, but when there is a resultant reaction, which tends to alter the cutaneous circulation or more especially to cause a sympathetic response, an indirect effect may be brought about. What evidence there is seems to point also to the view that nutritional deficiency does not affect the hair differentially to any appreciable degree.

In regard to endocrine factors, the evidence is very unsatisfactory, but there is little doubt that the internal secretions exert an important influence on the development of the hair. Those glands which, at present, seem most important are the suprarenals, the gonads, the thyroid and the pituitary, though in the last two instances it is possible that the effect is not direct, but that their disturbances react on the suprarenals which, in turn, influence the hair. The sexual dimorphism and the growth of increased amounts of terminal hair at puberty direct attention to the gonads, but there is no certain knowledge of their action. There are two diametrically opposed theories to account for these sexual differences: first, that the male type of hairiness is latent in all persons but that its development over the greater part of the body is suppressed by the action of the ovary; second, that the hairiness of the male is due to stimulation by testicular secretion, in the absence of which body hair remains undeveloped except within narrow limits. It is possible, on the basis of certain inferences drawn from clinical cases, that there may be a differential response by the hair in different parts of the body, with the result that testicular or some other hormone may be favorable to hair growth in one part and not in another. From clinical data, pointing to an association between hyperfunction of the cortex and excessive growth of hair, one would suspect that the suprarenal was the most important gland having an influence on the growth of hair.

To summarize, the growth of the hair is determined by the inherent character of the follicles, which are potentially different in different races, persons and parts of the body. There is considerable evidence suggestive of what some conditioning factors for hair growth are, but anything like a useful knowledge of their mode of action is lacking.

In the second part of this paper, the author discusses a number of aberrant types of hair growth. These fall into two categories: those due to infections

by parasites, and those, such as trichorrhexis, monilethrix and ringed hair, due to some direct or indirect influence of the vegetative nervous system. He does not consider that any true explanation has yet been advanced for the hair turning gray. In regard to alopecia, the author considers that some cases may be due to a modification of the subcutaneous tissues which have no muscular layer beneath them; in other cases, those appearing without obvious cause before the thirtieth year, he suspects that it may be a definitely hereditary, sex-limited trait, dominant in men, recessive in women. The third type, those not due to pathogenic organisms or to specific trophic disturbances, he considers for the most part hereditary and frequently congenital.

PEARSON, Philadelphia.

THE CHEMICAL COMPOSITION OF THE SPINAL FLUID. HERBERT B. WILCOX and JOHN D. LYTTLE, with the technical assistance of J. E. HEARN, Am. J. Dis. Child. **30**:513 (Oct.) 1925.

The purpose of this work was the determination of the general or selective permeability of the choroidal cells under the influence of disease. The differentiation of acutely inflammatory conditions and also of those less acutely inflammatory is both chemically and clinically confused. Bacteriologic diagnosis is positive when it can be determined. The permeability of the choroidal cells is probably affected in a varying degree by different types of physical disturbance and is not always equal for all elements entering into the composition of the blood. The chemical composition of the spinal fluid seems to be determined by three factors: the chemical content of the blood, by means of the permeability of the choroid plexus; mechanical agents causing congestion of the covering membranes; acute inflammatory conditions of the meninges, brain and cord. Complete knowledge of the chemistry of both blood and spinal fluid and also of the localization of the disease is necessary for an accurate interpretation of the results obtained.

Five groups were studied as follows:

GROUP 1.—Miscellaneous cases with supposedly normal spinal fluids. The chlorides of the spinal fluids averaged 723 mg. per one hundred cubic centimeters of fluid while the blood chlorides averaged 576. Spinal fluid protein averaged 52 mg. The percentage relation of the spinal fluid sugar to the blood sugar was from 40 to 60.

GROUP 2.—Cases of meningismus complicating pneumonia or other infections; tetany with convulsions; acidosis; nephritis. The chloride changes were irregular and not characteristic. High protein was reported in a few of each group of cases. A large percentage of the cases in the group showed a high sugar content both relatively and absolutely.

GROUP 3.—Cases of encephalitis and poliomyelitis. Spinal fluid chlorides were low in 36 per cent of the cases of poliomyelitis but in only 8 per cent of the cases of encephalitis. The protein was high in 45 per cent of both types of cases but showed a tendency to fall during the stage of clinical improvement. Sugar content was normal in poliomyelitis and high in encephalitis.

GROUP 4.—Cases of acute, purulent, chronic and syphilitic meningitis. Chlorides were low in purulent meningitis but normal in chronic and syphilitic meningitis. Protein was highest in the acute purulent type but increased in all three. Spinal fluid sugar was both absolutely and relatively reduced in the purulent group and showed a tendency to reduction in the syphilitic group.

GROUP 5.—Cases of tuberculous meningitis. All showed low spinal fluid chlorides; blood chlorides were also decreased. The protein was very much increased in all cases with a tendency to further increase as the disease progressed. Sugar was relatively low in all cases and actually low in most cases.

Calcium determinations were made in eighteen cases, five with tetany. The average for those with tetany was slightly lower both in blood and spinal fluid. Phosphorus content of the spinal fluid was found to be independent of blood phosphorus and to be increased in acute and tuberculous meningitis. Acetone determinations were made on blood and spinal fluid but the findings did not show its presence to be of diagnostic value. The methods used in making determinations were in accord with the newest accepted technic and in some cases the results were checked by other methods.

Three explanations are offered for changes in the composition of the spinal fluid in disease: changes in the composition of the blood; change in the permeability of the choroid plexus by disease or toxin; bacterial activity. No one of these three possible explanations will seem to account for all the changes found, although each will account for some. Chemical changes in the spinal fluid do not, as yet, seem clearly enough defined to be used in the differential diagnosis of the diseases cited.

WAGGONER, Philadelphia.

THE PUPILS IN SOMATIC AND VISCERAL DISORDERS IN ASSOCIATION WITH REFERRED PAIN AND HYPERALGESIA. JOSEPH BYRNE, J. Nerv. & Ment. Dis. **63**:105 (Feb.) 1926.

Lack of knowledge of normal pupillary reactions to pain and tenderness in somatic and visceral disease has been felt in medical diagnosis. The author on the basis of previous work declares that pupil dilatation is effected mainly by the pain-bearing or affective system, whereas pupil constriction is effected by the critical or proprioceptive system. An inherent preponderance of constrictor tonus keeps the pupils contracted in sleep. Normally there exists a balance between the dilator and constrictor impulses which regulates the pupillary size and equality. After lesions of the peripheral somatic nerves two phenomena occur, a true paradoxic dilatation and a pseudo-paradoxic dilatation, both occurring in the homolateral pupil after unilateral lesions of the nerves above the level of the umbilicus, or in the contralateral pupil after lesions below that level. The pseudo-paradoxic phenomenon occurs twenty-four hours after the lesion, whereas the true paradoxic dilatation occurs from the eighth to the fifteenth day after and is of less clinical importance. Immediately after the injury to a nerve the contralateral pupil may be the larger, but when the effect of the injury stimulus has passed off the contralateral is found to be the smaller pupil if the animal has been left absolutely quiet. If, however, he is excited or hurt the contralateral pupil becomes at once the larger. This then is the pseudo-paradoxic phenomenon. The constriction of the contralateral pupil when the animal is quiet is accounted for by diminished inflow of dilator or affective impulses resulting from functional impairment of the primary affective neurons. The dilatation on excitement or pain results from overdischarge of the less seriously injured primary affective neurons. That which determines which eye will exhibit the pseudo-paradoxic phenomena is the development of the viscera from right or left side of the primitive gut, and their nerve supply arising from either right or left side of the spinal cord. The pseudo-paradoxic phenomenon occurs in the pupil homolateral to the

visceral lesion when the organ receives its afferent supply from segments above the tenth thoracic and occurs in the contralateral eye when the nerve supply is below that level. Lesions involving liver, gallbladder, pancreas, or parietal peritoneum gave doubtful or negative results. The author illustrates the phenomenon in eighteen selected cases of unilateral somatic and visceral disease, above and below the level of the umbilicus. The author believes that the dilatation phase of the pupillary reaction is due to hyperfunctioning of the primary affective neuron, because when produced by section of one sciatic nerve, it completely disappeared on section of the spinal cord in the upper lumbar region. The author finds that deep hyperalgesic areas usually appear in visceral disease before the superficial and are often elicited only by deep pinching or deep digital pressure. Reflected pain is often found in the vicinity of an area of deep hyperalgesia. In order to produce the phenomenon the visceral lesion must be completely or preponderantly onesided. In most cases of heart disease the ocular phenomena occurs in the left eye. In kidney, testis, ovary or fallopian tubes the secretory portion usually derives its nerve supply from the spinal cord above the tenth thoracic segment, while the duct system is supplied by nerves entering the cord below that segment. Here the pupillary phenomenon occurs in either eye, according to which system is involved. The author concludes that the phenomenon is a reliable physical sign of somatic lesions associated with pain and tenderness, and when taken with referred pain, forms a reliable corroborative localizing sign of visceral disease. It may be useful in the detection of malingerers. He believes the areas of reflected pain and hyperalgesia described by Head require revision, and also that the study of the pupils in association with referred-reflected pain and hyperalgesia should be part of all routine physical examinations.

HART, Philadelphia.

STUDIES ON MUSCLE TONUS. I. CONTRACTILE AND PLASTIC FACTORS IN DECEREBRATE RIGIDITY. II. A COMPARISON OF THE SYNAPSE-BLOCKING ACTION OF NICOTINE AND CHLORAL HYDRATE. III. SUBLAMINAL INJECTION OF CHLORAL HYDRATE IN DECEREBRATED CATS. S. W. RANSON, J. Comp. Neurol. **40**:1 (Feb.) 1926.

A new approach to the problem of tonus mechanisms is opened in these studies. Taking his departure from the spinal reflex tonus arc of Sherrington ("antigravity reflex"), the author calls attention to the fact that cutting the dorsal spinal roots breaks the proprioceptive arc and so blocks investigation of some aspects of the question at the start. The application of nicotine to the spinal ganglia and dorsal roots of decerebrated cats, on the other hand, blocks the tonic impulses without interfering with sensory conduction. The stiffness of the leg on the treated side is abolished by five or ten minutes' application of a 0.04 per cent solution of nicotine, but experiment shows that the proprioceptive reflex arc responsible for the extensor thrust is still functional. Indeed the latter may be preserved after twenty minutes' application of 0.24 per cent nicotine.

The inference drawn from the experiments is that the synapses known to occur within the spinal ganglion are affected by the drug and that plastic tonus is thereupon abolished without impairment of other forms of nervous conduction. The source of the fibers ending in these synapses has not been determined. Apparently they do not come in by way of the sympathetic nervous system. The application of nicotine to the spinal ganglia does not make the corresponding limb flaccid. A certain amount of tonus, apparently identical

with the contractile tonus of Langelaan, remains. Only plastic tonus is lost. The theory is advanced that plastic tonus is maintained by nerve impulses which leave the spinal cord through the dorsal roots and pass through synapses in the spinal ganglia and then outward by antidromic conduction in the peripheral fibers of the spinal ganglion cells.

In the second study, after the previous experiments with nicotine were repeated and various controls applied, the synapse-blocking action of solutions of nicotine and chloral hydrate were compared by applying them directly to the superior cervical sympathetic ganglion. Dilutions of from 0.05 to 0.03 per cent nicotine and 1 to 2 per cent chloral hydrate block the superior cervical ganglion for pupillodilator impulses. Nerve fibers are less easily damaged than synapses, and a 5 per cent solution of chloral hydrate, acting on the superior cervical ganglion, did not alter the conductivity of the postganglionic fibers. Chloral hydrate is less toxic than nicotine and it does not act as a medullary stimulant. It is therefore to be preferred in further study of the relations of synapses in spinal ganglia to plastic tonus.

In the third study a method is described of sublaminal injection of chloral hydrate without performing a mutilating laminectomy. Fluid can be easily injected into the space between the dura and the wall of the spinal canal within which it becomes widely distributed and comes in contact with the spinal ganglia. The injection beneath the lamina of the seventh lumbar vertebra of a few cubic centimeters of a 1 or 2 per cent solution of chloral hydrate produces in decerebrated cats a marked decrease in the rigidity of the hind legs without any impairment of the spinal reflexes. Such small doses never entirely abolish the muscle tonus; the legs do not become flaccid. The injection of much larger amounts of chloral hydrate is required to abolish muscle tonus completely, and when the legs become flaccid the spinal reflexes are lost at the same time.

These results lend support to the hypothesis that muscle tonus is composed of two factors: contractile tonus due to nerve impulses reaching the muscle through the motor fibers of the ventral root, and plastic tonus due to nerve impulses which pass through synapses in the spinal ganglia. The reduction of the rigidity in decerebrated cats brought about by the sublaminal injection of weak solutions of chloral hydrate is probably due to the blocking of these synapses in the spinal ganglia.

HERRICK, Chicago.

THE INTERPRETATION OF THE ELECTROMYOGRAM OF STRIATED MUSCLE DURING CONTRACTIONS SET UP BY CENTRAL NERVOUS EXCITATION. J. G. DUSSER DE BARENNE and J. F. G. BREVÉE, J. Physiol. **61**:81-97 (March 18) 1926.

Thirty-eight experiments were made on the decapitate cat and a few on the spinal frog. The dorsal surface of the spinal cord was exposed throughout the cervical region. Silver needle electrodes coated with silver chloride were thrust into the muscle (triceps brachii). Action currents were recorded with the large model Edelmann galvanometer with 3.5 amperes through its electromagnets and a gold string of 2 microns diameter which follows vibrations up to 660 a second. In some experiments the posterior roots were cut in the cervical region. In all experiments the cord was painted with novocaine. The stimulus employed was a light touch to the cord. Records were taken prior to the application of novocaine and at various intervals thereafter.

The action currents prior to narcosis resemble those obtained in voluntary movement: the oscillations recorded are irregular in size and frequency. The

frequency varies from 100 to 200. The frequency is the same for different strengths of contraction; the amplitude is greater the stronger the contraction. After local narcosis the frequency is reduced gradually to from 50 to 70 a second and the rhythm sometimes becomes regular.

The authors draw two conclusions: 1. These slow and regular action currents represent the "proper" rhythm of the impulses of the motor spinal mechanisms, of the motor neurons free from the various centripetal impulses which play normally on the neurons and give rise to a change of the slow, regular, proper rhythm into the frequent and quite irregular excitations shown by the normal electromyogram. 2. The electromyogram of striped muscle during contractions of central origin is a true image of the central impulses, reaching the muscles from the central nervous system along the peripheral nerve fibers.

The conclusion that the final regular rhythm is the "proper" rhythm of the anterior horn cells implies that these cells still function normally when this rhythm is obtained. Were this the case, we should expect a considerable period during which contractions remained maximal and string oscillations were at least equal in amplitude to the maximal oscillations of the normal electromyogram, since the interval between oscillations exceeds the relative refractory period of cat muscle at body temperature. Actually, the contractions become smaller as the narcosis progresses. In the four series of records produced, the maximal amplitude of oscillations in the record showing regular rhythm is far less than that of the record preceding narcosis. In three cases out of four, the oscillations are so small as to suggest that the response is limited to the small group of muscle fibers innervated by a single anterior horn cell after the elimination of conduction in all neurons responding out of phase with it. The picture appears to be one of a progressive narcosis, eliminating anterior horn cells from activity seriatim. One of the early effects of narcosis is slowing of the recovery process—lengthening of the refractory period. The authors are inclined, therefore, to regard the final rhythm obtained in these experiments as a measure of the prolongation of the refractory period of a partially narcotized anterior horn cell. Under these conditions, the rhythm in the muscle is undoubtedly that of the nerve. These experiments present no evidence that such a relation obtains in the case of the normal electromyogram.

McCouch, Philadelphia.

Dermatitis in Association with Disease or Injury of the Peripheral Nerves. S. William Becker, Arch. Dermat. & Syph. **12**:235 (Aug.) 1925.

Becker reports the case of a woman, aged 47, who developed a distinct erythema and papulo-vesiculo-pustular eruption on the right side of her face three weeks after section of the posterior root of the right trigeminal nerve for bilateral facial neuralgia. The neuralgia had been present for thirteen years on the left side and for eight years on the right, and treatment of both sides by alcohol and osmic acid injections had been ineffectual. The eruption had existed unchanged for nine months, except for exacerbations at the menstrual periods, when she was seen by Becker, and continued for the next two years despite local treatment. At the time of the first examination, there was complete anesthesia of the right side of the scalp and face, including the oral mucosa, and the facial condition was accompanied by a seborrheic dermatitis, which led to the diagnosis of seborrheic dermatitis with a probable trophic element. When examined two years later, the same condition was found with the addition of marked fetor oris and caries of the teeth of the right side.

Roentgen-ray treatment was commenced and produced amelioration of the facial dermatitis, but, up to the time of the report, the improvement was maintained only by the constant use of local applications.

A review of the literature shows that dermatitis having an apparently intimate connection with a derangement of the nervous system manifested by a history of trauma and by confirmatory signs and symptoms or merely by neuralgia and neuritis, seems to have been either extremely rare or else overshadowed by the more apparent nerve injury and so not considered worthy of mention. The detailed reports of 107 cases of removal of the gasserian ganglion or section of the posterior root, taken at random from the English, French and German literature of the last thirty years, revealed no simliar case, and cutaneous alterations were mentioned in only five cases.

A number of theories have been evolved to explain eruptive disturbances of a nutritional or trophic nature. These are summarized best in the table quoted from Leloir by the author:

A. Nonnervous theories: (1) influence of external agents (compressions, etc.); (2) influence of internal agents (bacteria, toxins, etc.); (3) functional inactivity; (4) propagation of inflammation along the nerves to the tissues supplied by them.

B. Nervous theories: (1) vasomotor: (*a*) vasodilator (neuroparalytic hyperemia); (*b*) vasoconstrictor (neurogenous ischemia); (2) direct trophic disturbance: (*a*) direct irritation; (*b*) trophic nerves; (*c*) diminution or absence of trophic action; (3) reflex disturbances: (*a*) irritation of the centers by centripetal excitation; (*b*) abolition or diminution of action of the centers by centripetal excitation.

The author's review of the literature concerning the etiology shows as much weight of authority behind one theory as behind another. Bruce's work appears to make one thing certain. The immediate result of section of the posterior root is not a reflex inflammation, for he found that section of the root proximal to the ganglion produced no change in the condition of the skin; but anesthetization or section of the root distal to the ganglion inhibited the appearance of inflammation when irritants were applied to the skin.

PEARSON, Philadelphia.

THE MORPHOLOGY OF THE GASSERIAN GANGLION. CHARLES H. FRAZIER and EDWARD WHITEHEAD, Brain **48**:458 (Dec.) 1925.

Section of the sensory root of the fifth nerve for major trigeminal neuralgia has been performed by Frazier 396 times since 1901 with a mortality of 0.5 per cent. In the course of time the operation has led to various changes: leaving intact the motor and cutting the sensory root only, and subtotal resection whereby the two inner fasiculi of the sensory root are spared. Of twenty-five selected cases in which operation was performed by the latter method not one developed keratitis, nor was there any recurrence of pain in any of the three divisions of the trigeminal nerve. A morphologic study of the gasserian ganglion in human embryos, aged from 4 to 28 weeks, was subsequently undertaken by serial sections, wax models and Cajal stains being employed, and in the older embryos by dissection. From the fourth week to term the semilunar ganglion is never separated into three distinct portions corresponding to the three nerve divisions, but is a single fused mass. Though never a completely detached unit, the ophthalmic division is differentiated from the rest of the ganglion by a more precocious development in that its cells develop their fiber processes earlier and become arranged in clusters sooner than do

the cells of other regions, and likewise the ophthalmic ventral cellular process, the precursor of the ophthalmic nerve, develops earlier than do the maxillary and mandibular processes; the site of formation of the ophthalmic nerve is widely separated from the other two branches which are close together. Owing to the fact that the proliferating cells of the ganglion, which was at first an oval mass, tend to split in the axis of the cell processes, the increase in size of the ganglion is in a plane transverse to the main axis of the nerve; as its growth increases it becomes semilunar in shape and is characterized by a hilum from which the sensory root emerges. The peripheral processes of the ganglion cells reach the nearest point on the surface of the ganglion, then pass along the surface in an orderly manner and become collected in the nearest of the three trunks; the fibers of the central root run parallel and become arranged in separate bundles by the enveloping supporting tissue and do not tend to intermingle. By following the ganglion development and the course of its processes, it can be definitely stated that a cell located in any given part of the ganglion sends its proximal process upward into a corresponding part of the root and its peripheral process out into the corresponding peripheral nerve. While in its early stages the motor root emerges from the pons ventral to the entering sensory root, in later stages as the ganglion rotates the motor root emerges cephalad to the point of entrance of the sensory root. The motor root emerges from the pons as separate bundles at two different levels, and the fibers emerging from the dorsal level are apparently of the mesencephalic nucleus; as it passes across the median surface of the ganglion, it is joined by peripheral sensory fibers that have arisen from sensory cells in the maxillary region of the ganglion.

STACK, Milwaukee.

THE ASYMMETRY OF THE HEMISPHERES OF THE BRAIN IN MAN AND ANIMALS. T. RASDOLSKY, J. Nerv. & Ment. Dis. **62**:119 (Aug.) 1925.

The asymmetric development of man's cerebral hemispheres, constituting a new phase in the evolution of the vertebrate nervous system, is associated with two functions, the gnostic and the praxic, the gnostic functions constituting the basis of the praxic. The degree, however, of anatomic asymmetry does not correspond with the functional distinctions that we observe, and the reason is probably that morphologic asymmetry rests not so much on gross anatomic alterations as in differences in degree of the finer histologic organization. Asymmetry of function of the human cerebral hemispheres is innate and in no way a matter of choice or imitation. Left-handedness like right-handedness is hereditary. Fleschsig found that the left visual tract myelinized before the right. Theories of causation of this asymmetry based on difference of arterial blood supply or in acuity of vision seem inadequate, while Weber's declaration that writing is the only agent which created the asymmetry does not explain all the facts. In civilized man asymmetry of the hemispheres is greater than in the savage, and in the individual asymmetry of function is first shown from the sixth to eighth month of life in motor unidexterity. Speech, writing and reading functions are lateralized later. In the author's opinion the cause of asymmetry lies in the formation of conditioned reflexes only in one hemisphere, and probably began with the assumption of the erect posture when man in using his prehensile upper extremities came to employ one more than the other. The primitive man must have observed that repeated performance of a function with the same hand facilitated him in many actions and, later,

especially in the art of writing. As contributory influence toward right handedness the author cites the left sidedness of the heart and the fact observed by Mackenzie that movements of the left hand reflect more strongly on the work of the heart. The preeminent use of the right hand in writing led to the corresponding function developing only in the left hemisphere and the author believes that through neurobiotaxis an attraction was exerted in the rest of the functions, lateralizing them to that hemisphere. Primitive man gesticulated more in his speech at first and doubtless used the right hand more than the left, leading probably to the development of the speech center in the left hemisphere. The influence of the gnostic or sensory perceptional functions of the brain being served bilaterally tends to promote the symmetric organization of the hemispheres, thus contrasting with the asymmetric influence of the praxic functions. The author is convinced that asymmetry will persist as long as the functional specialization of the hands persists.

HART, Philadelphia.

THE VIRUS OF HERPES; ITS IMMUNE REACTIONS AND ITS RELATION TO ENCEPHALITIS LETHARGICA. J. R. PERDRAU, Brit. J. Exper. Path. **6**:41-52 (April) 1925.

The work here described is a continuation of that reported by Parsons, McNally and Perdrau in 1922. Perdrau ruled out the possibility of spontaneous encephalitis in his experimental animals; it was necessary for him to obtain a new strain of virus as those previously used had died out. The new strain was obtained from twenty-four hour vesicles on the lip of a woman with an ordinary coryza. Intracerebral inoculation of passage virus from known epidemic encephalitis was fatal in thirty-nine inoculated cases, and the herpes virus, given in the same manner, caused positive results in six of seven animals. Recovery after onset of symptoms pointing to encephalitis, the result of intracerebral inoculation, was never observed. Recovery did take place in four instances in which other portals of entry were used. Inoculation by the corneal route resulted in kerato-conjunctivitis followed by an encephalitis in one case, with completely negative results in seven instances. When passage virus was rubbed onto the nasal mucous membrane with sufficient violence to cause scarification, seven of ten animals developed a typical encephalitis. When virus was injected intraperitoneally, two types of reaction were obtained depending on the strain (neurotropic or dermotropic) used. However, the dermotropic strain acquired neurotropic properties with immunizing powers by a single cerebral passage and the neurotropic strain lost its neurotropism by second passage. The clinical picture in the fatal cases was characterized by lethargy, photophobia and slight restlessness but no convulsions. The histologic lesions were usually slight and in some cases barely recognizable. However, the author feels that the results of intracerebral inoculations of emulsions of the brains of fatal cases of epidemic encephalitis was identical with those obtained in animals which succumbed to an intracerebral inoculation of that virus. He concludes that cerebral immunity to the virus of herpes is best obtained by intradermal inoculations of the neurotropic virus. This immunity is fully established by the eleventh day and affords protection for about three months. He believes further that this immunity is essentially of a cellular nature. The inoculations must be carefully done as a true encephalitis may result if scarification is at all deep.

POTTER, Akron, O.

THE INCUBATION PERIOD OF BENIGN TERTIAN MALARIA. G. DE M. RUDOLF, J. Ment. Sc. **72**:69 (Jan.) 1926.

The article is especially well summed up in the words of its author: "A series of fifty general paralytics inoculated subcutaneously with *Plasmodium vivax* showed that 90 per cent gave a rise of temperature of from 101 to 102.9 F. in less than twenty-one days after inoculation, and 46 per cent in less than ten days. The first rise of temperature over 103 F. occurred within twenty-one days in 84 per cent and within ten days in 38 per cent.

"Following subcutaneous inoculation there is a well-marked correlation between the first rises of temperature to 101 F. and to 103 F. and the first finding of parasites in thin films. The frequency-curves of the first finding of parasites in thick films and of the first rise of temperature to 101 F. are very similar, although the observations were made on two different series of cases inoculated with two different strains of parasite. Curves of the first finding of parasites in thin films and the first rise of temperature to 103 F., in the same series of cases, are also somewhat similar.

"Intravenous inoculation of malarial blood gave an incubation period of from one to nineteen days in a series of sixty cases collected from the literature.

"In a series of twenty-eight cases inoculated intramuscularly at Bexley and Winwick Mental Hospitals the incubation period varied from six to twenty-three days.

"In ten series of cases injected subcutaneously with malarial blood it was found (*a*) that when similar numbers of parasites were injected the incubation periods were of somewhat similar lengths, and (*b*) that when different numbers of parasites were injected the incubation periods showed a marked tendency to be shortest when the dosage of parasites was the greatest. In one series of four cases this relationship did not hold as regards the first rises of temperature, but only as regards the first dates on which parasites were found. In most series the length of the incubation period as measured by the first time that parasites were found, under standard conditions, corresponded more nearly to the dosage than did the length of the period as measured by the first rises of temperature."

BOND, Philadelphia.

SPINAL HYPERTROPHIC PACHYMENINGITIS AND MEDULLARY CAVITIES. EGAS MONIZ, Rev. neurol. **2**:433 (Oct.) 1925.

The author does not agree with Philippe Oberthür concerning the classification of intramedullary cavities, and discusses the existence of a syringomyelic pachymeningitis in which the syringomyelic cavities are supposed to be secondary to the pachymeningitis. He is inclined to regard spinal hypertrophic pachymeningitis as an autonomous disease in which the thickening of the meninges produces vascular changes which are the cause of cavity formation. Such autonomy, first described by Charcot and Joffroy, has been confirmed experimentally by Camus and Roussy, and by Lhermitte and Boveri. On the other hand, the histology of the medullary cavities in pachymeningitis is somewhat different from that of the cavities in syringomyelia. The ependymal canal takes no part, at least to any great extent, in the formation of pachymeningitis cavities. The general appearance is different in the two conditions: the contours are irregular in pachymeningitis; the neuroglial reaction is much more marked in syringomyelia. Lastly, the cavities in pachymeningitis frequently affect the white matter.

The author then describes three types of pachymeningitis: tuberculous, syphilitic and one of unknown cause. In cases of pachymeningitis of tuberculous and syphilitic origin the almost constant presence of meningomyelitis due to inflammatory vascular changes renders the tissue less resistant, so that the cavity formation has neither the diffusion nor the characters of the typical cavity; sometimes, even, cavities are absent. When the spinal parenchyma is not affected, the results of compression are more important because of the resistance of the medullary tissue to the meningeal strangulation. The circulatory disturbances that follow explain the cavity formation.

The conclusions reached are: Hypertrophic pachymeningitis of nonspecific nature is the form most connected with cavity formation, and the type of cavity differs from that of syringomyelia. From the therapeutic point of view, surgery, which is often open to discussion in cases of specific nature, is to be recommended in pachymeningitis of unknown origin, especially when localized.

FERRARO, Washington, D. C.

SPINAL FLUID FINDINGS IN PEMPHIGUS AND IN DERMATITIS HERPETIFORMIS. ALBERT STRICKLER and CLAUDE P. BROWN, Arch. Dermat. & Syph. **12**:48 (July) 1925.

The authors studied the cerebrospinal fluid in three cases of pemphigus and in two of dermatitis herpetiformis and report one case of the former in which the cell count was increased, one case that showed a positive globulin reaction and a positive sugar reduction reaction, and one case in which the fluid was normal. In the latter condition, one case showed a borderline pleocytosis, and the other an increase in cells and a positive globulin reaction. In each of the five cases cultures of the cerebrospinal fluid were negative.

The majority of necropsy reports on cases of pemphigus in the literature appear to indicate that the pathologic picture in this condition is one of chronic inflammatory changes in the dorsal roots, in the root ganglions, in the dorsal columns and in the dorsal horns of the gray matter of the spinal cord. Strickler and Brown conclude from these reports and from the cerebrospinal fluid findings in their own cases that pemphigus bears an analogy to both tabes and herpes zoster in that the skin lesion is a consequence of the involvement of the dorsal roots. The agent responsible for the changes in the dorsal roots is of the nature of a subacute or chronic toxemia. At times this toxemia is complicated by secondary infections, such as those produced by *Bacillus pemphigus* (of Eberson), *Bacillus pyocyaneus* and others, which enter the blood stream and further devitalize the patient's resistance and, in a large measure, are responsible for the frequently fatal outcome of the disease.

They advocate intravenous injections of sodium cacodylate in doses of from 3 to 5 Gm., given every other day or three times a week, and massive doses of phenobarbital by mouth, in the treatment of pemphigus.

Though the authors draw no conclusions as to the relationship between pemphigus and dermatitis herpetiformis, the results of the cerebrospinal fluid examinations in the latter condition would lead to the conclusion that there is a possible relationship of the two diseases.

PEARSON, Philadelphia.

A SEVERE FORM OF MIGRAINE WITH ANATOMIC FINDINGS. PAUL HILPERT, Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:478 (July) 1925.

Hilpert reports a case of severe migraine in a man, aged 60. There was a distinct family history of migraine. The grandmother, mother and sister of

the patient had severe attacks of migraine with aphasia, hemiplegia, disturbances of vision and even disturbances in consciousness. The patient had motor and sensory paralysis on one side, complete aphasia with alexia, agraphia and apraxia, and hemianopia. Hallucinations and rapidly progressing dementia were features of the case. Bradycardia and fever showed the presence of vegetative involvement. Schob who reported the case previously, thought that the vertigo and ataxia were due to involvement of the cerebellum, but necropsy revealed no lesion in the cerebellum. The author attempts to account for the severe headache. Quincke assumed that the headache in migraine followed as a result of increased pressure due to a serous exudation in the subarachnoid spaces, a sort of angioneurotic hydrocephalus. He reported on the beneficial results of lumbar puncture in migraine. Others, as Lennander, believe the headache is due to stretching of the dura in increased intracranial pressure, and still others look on it as due to angiospasm. Stöhr has recently shown that the nerves of the pia and of the pial sheaths around the vessels are derived from the carotid and vertebral plexuses and from branches from the third, sixth, ninth, tenth, eleventh and twelfth cranial nerves and that they end in small end-corpuscles. In view of this, the headaches become explainable from transudation in the pial sheaths producing a local effect, or from an acute increase in the cerebrospinal fluid which is not reabsorbed as quickly as it should be. In the same way this production of hydrocephalus would explain the vomiting as due to irritation of the ending of the vagus nerves in the pial sheaths.

Necropsy showed no gross abnormalities. Microscopically, there was fatty degeneration in the entire cortex, affecting chiefly the fifth layer especially in the temporal lobes. The glia cells contained much fat. The basal ganglia, midbrain and cerebellum showed little fat. Corpora amylacea in great number were found in the walls of the lateral ventricles, in the optic sheaths and in the cornu ammonis. They were present also in the occipital cortex.

ALPERS, Philadelphia.

PHRENIC NERVE INJURY IN THE NEW-BORN. E. FRIEDMAN and R. S. CHAMBERLAIN, J. A. M. A. **86**:934 (March 27) 1926.

After a review of the literature and discussion of a case of diaphragmatic palsy due to phrenic nerve injury at birth reported by Kofferath, the authors stress the following points: 1. A diagnosis of phrenic nerve injury can be made with certainty only by the roentgen ray. Other conditions present, in addition, the following: pneumonia, fever and leukocytosis; persistent thymus, stridor; pneumothorax, unilateral hyperresonance; cerebral injury, abnormal spinal fluid. Atelectasis and diaphragmatic hernia also present characteristic signs. 2. Return of function is gradual and in case of bilateral phrenic palsy the outlook is grave. 3. The incidence of birth injuries may be reduced by refraining from injudicious intervention with the progress of labor. 4. Treatment of the arm should be instituted and the orthopedist and physiotherapist called into action.

Believing phrenic nerve injury to be a not uncommon birth complication, the authors report a case which is briefly that of a girl who shortly after normal birth with low forceps showed paralysis of the right arm. "At the age of 13 days, respirations became hurried and irregular and occasional cyanosis appeared. The brachial paralysis disappeared in two weeks. Breathing was fast and irregular for three months and at the age of 8½ months she enjoyed perfect health." Recognition of the condition was accidental and effected by the roentgen ray.

The authors conclude that phrenic nerve palsy is not a rare type of birth injury and that its presence should be excluded in every case of brachial palsy; the same obligation devolves on the physician when the baby displays cyanosis, irregular or accelerated respirations, unilateral diminution of breath sounds or serious digestive difficulties.

CHAMBERS, Syracuse, N. Y.

AMOUNT OF CEREBROSPINAL FLUID IN HYDROCEPHALUS. METHOD FOR ESTIMATION AND ITS CLINICAL APPLICATION. A. GRAEME MITCHELL and MENDEL ZELIGS, Am. J. Dis. Child. **30**:189 (Aug.) 1925.

Because of the difficulty in determining the progress of hydrocephalus, the authors have devised a method by means of which the amount of cerebrospinal fluid can be determined. The amount of fluid can be used as an index to the progress of the disease. First, the type of hydrocephalus is determined; i. e., obstructive or communicating, and if communicating, the degree. This is done by injecting neutral phenolsulphonphthlein into the ventricle and determining the length of time necessary for it to appear in the spinal canal as determined by lumbar puncture (Dandy and Blackfan). To determine the amount of fluid in the ventricles, 1 cc. of neutral phenolsulphonphthalein is injected into the ventricle by intraventricular puncture. The syringe is washed free of dye by withdrawing and reinjecting the ventricular fluid several times before withdrawing the needle. The head is then raised, lowered and rotated from side to side several times. Fifteen minutes later some of the fluid is removed and the percentage of dye contained is determined by the colorimetric method. If the case is one of communicating hydrocephalus the amount of dye excreted during the first half hour after injection is ascertained and allowance is made for this in the final result. Errors may result from: imperfect admixture of dye with the cerebrospinal fluid; blocking off of some part of the cerebrospinal system by adhesions; failure accurately to measure the dye.

The authors cite a case in which this method was used and show that the findings follow the progress of the disease. At necropsy, the amount of fluid found and the amount estimated by the authors' method checked fairly well. The method was useful in judging the progress of the disease in this case.

WAGGONER, Philadelphia.

THE WORK OF SIR CHARLES BELL IN RELATION TO MODERN NEUROLOGY. H CAMPBELL THOMSON, Brain **48**:449 (Dec.) 1925.

Sir Charles Bell was a leading anatomist and one of the first to apply physiology to anatomic structure, making many discoveries in the structure and function of the nervous system. His investigations led to the discovery of the differing functions of the two roots of the spinal nerves and their division into a motor and sensory portion. This theory he likewise applied to the two divisions of the fifth cranial nerve in the innervation of the face. The formulation of his "Respiratory System of Nerves", though modified later, drew attention to the importance of muscular movements in respiration, which until the time was thought by the physiologist to be wrought by certain chemical changes in the lungs. He likewise recognized the difference between the bilateral action of certain muscles for involuntary movements and unilateral action for voluntary movements. Among his other works was the verification of nerves to muscles in which he states, "between the brain and the muscles there is a circle of nerves: one nerve conveys the influence from the brain to the muscle, another gives the sense of the condition of the muscle to the

brain". Bell's recognition of different levels of function, in which he states that additional superadded nerves to the original system (lower form of animals) do not destroy but only obscure that system, gave rise later to the important principle laid down by Hughlings Jackson that destructive lesions produce negative conditions which permit positive symptoms to appear. By applying a weight to the extensor muscle and then observing it descend while the flexor muscle contracted, Bell was one of the first to recognize the idea of reciprocal innervation of muscles.

STACK, Milwaukee.

EYEDNESS AND HANDEDNESS. LLOYD MILLS, Am. J. Ophth. **8**:933 (Dec.) 1925.

True ambidexterity is rare and is considered atavistic. Dextrality is the dominant condition in man, although left-handedness as a familial tendency is found in not less than 20 per cent of all people, which bears out the explanation that it persists as a mendelian recessive. The eyes share in the bilateral asymmetry of the structure and function of the paired organs of the human body. The right eye usually possesses a greater sense of clarity and power of discrimination than the left. There are four groups of dextrals and sinistrals: The pure dextral is right-handed and the right eye is the master eye — about 76 per cent; the pure sinistral is left-handed and the left eye is the master eye — about 9.3 per cent; the crossed dextral is right-handed (usually by training) but is left-eyed — about 13 per cent; the crossed sinistral is left-handed but the right eye fixes — about 1.7 per cent. Ocular mastery determines handedness. The master eye regulates the matter of dominance of the corresponding side of the body which it guards and controls. Ocular mastery is determined by the use of the plane ophthalmoscopic mirror. The dominant eye maintains fixation inside the convergence near point. In doubtful cases the Maddox rod, rotary prism and Stevens clinoscope are made use of in determining the cyclophoric eye which is nearly always the nonfixing eye. Anisometropia plays relatively little part in causing ocular dominance. Moreover, if the pathways which produce ocular dominance are fully established, mastery may persist in spite of great loss of vision in the master eye. Ocular mastery should be maintained with care in the correction of errors of refraction since giving the mastery to the nondominant eye is likely to cause anguish to both patient and physician.

SCHUMACHER, Philadelphia.

ELECTRODIAGNOSIS AND ELECTROTHERAPY IN PARALYSIS FOLLOWING POLIOMYELITIS. RICHARD KOVACS, J. A. M. A. **86**:741 (March 13) 1926.

The author reports the methods of muscle testing and treatment by electricity as employed in the physical therapy department of the Reconstruction Hospital in New York. After the clinical staff has completed a thorough examination, the physical therapy department conducts tests for diagnosis and prognosis, and then institutes treatment. Kovacs then describes electrodiagnosis as applied to muscles, prognosis, rationale of electrical treatment and technic of electrical treatment. He says, "muscles responding to 0.09 microfarad of a condenser set will recover shortly; those with a response under 0.90 will recover under electrical treatment and orthopedic support. Those with a response over 1 microfarad still present a possibility of recovery." He believes that orthopedists have been indifferent or antagonistic because so little attention is given to electrotherapy in the medical curriculum and because treatments with which they may have had experience have been badly applied. In describing technic he emphasizes the

following points: treatment en masse is not as desirable as stimulation of individual motor points; overexercise of muscles should be avoided; electrotherapy needs well trained personnel; muscle training and reeducation should go on simultaneously with electrical stimulation.

Three cases from a total of forty-two are reported, two with full charts of the electrical tests. Kovacs concludes that: electrical currents simulating the natural response are most valuable in poliomyelitis; no electrical treatment should be begun without a previous accurate electrical test, and no case is of too long standing to preclude electrotherapy.

CHAMBERS, Syracuse, N. Y.

EPIDEMIC ENCEPHALITIS SEQUELAE AND THE PSYCHONEUROSES. AUGUST E. WITZEL, State Hosp. Quart. **10**:387 (May) 1925.

In reviewing the literature the author finds that very few patients with epidemic encephalitis are left without some sequelae, the most common being the parkinsonian syndrome and next cranial nerve involvement.. Sequelae may also resemble dementia praecox, general paralysis, psychoneurosis, multiple sclerosis and brain tumor. The author believes that two or more of the following symptoms are suggestive of the disease: fever, acute mental disturbance followed by lethargy, parkinsonian symptoms, myoclonic movements, especially of the abdominal muscles, and ocular palsies.

In the second part of the article the cases of four patients, aged from 8 to 17, were cited that had originally been diagnosed as psychoneuroses. Three of the four had a history of a febrile illness, influenzal in character, followed by conduct disorders, respiratory peculiarities, tics, tremors or choreiform movements. The fourth patient although not having the acute febrile illness presented the same clinical picture as the other three. Psychanalytic investigation disclosed the fact that two female patients had sex experiences of an unpleasant nature and the fourth, a boy, aged 17, had masturbated for several years. An association was found between the respiratory peculiarities and a similar type of breathing that occurred during the sex experiences. The author concludes that while at least three of the patients should be classed as having had epidemic encephalitis the twitchings, conduct disorders and respiratory disorders are just as likely to have a psychologic as an organic origin.

HOWARD, Milwaukee.

ATYPICAL AND MASKED FORMS OF ENCEPHALITIS. AXEL V. NEEL, J. Nerv. & Ment. Dis. **63**:1 (Jan.) 1926.

The author seeks to find certain pathologic changes in the spinal fluid in indistinct and atypical cases of encephalitis which have been diagnosed otherwise. He discovered that after 1921 certain changes in the spinal fluid occurred more frequently than before—very slight increase of albumin with or without a very slight increase of cells. Reports on spinal fluids of such cases between the years 1916 and 1921 were reexamined, and the presence of these findings confirmed the author's assumption that epidemic encephalitis occurred in Copenhagen during the years immediately preceding the announcement of the epidemics in Australia, France, Vienna and England. Of the 125 cases studied, pathologic changes were found in all but two cases, contradicting statements that spinal fluids in encephalitis show nothing abnormal. One case is reported of typical acute epidemic encephalitis resulting in death in which the spinal fluid appeared to be normal. Microscopic examinations revealed the following changes:

pronounced round cell infiltration around vessels, most noticeable in the central ganglia, cerebral trunk and cerebellum; pigment cells and small hemorrhages, perivascular in distribution, together with hyaline and other alterations in the vessel wall leading in some instances to obliteration. The vessel endothelium sometimes showed sufficient proliferation to produce this result. These changes were pronounced in sites usually affected by encephalitis in addition to the usual microscopic findings of encephalitis.

HART, Philadelphia.

THROMBOSIS OF THE SIGMOID SINUS FOLLOWING SKULL FRACTURE. O. JASON DIXON, Arch. Otolaryng. **3**:57 (Jan.) 1926.

Dec. 6, 1924, a man, aged 27, was brought unconscious to the hospital with a stellate fracture of the posterior parietal and occipital bones of the left side of the head. He regained consciousness, appearing to be improved until the fifth day when he complained of severe pain in his left ear and the left anterior side of his neck over the course of the internal jugular vein. There was no bleeding from the ears or nose at the time of the injury. The patient had never had any ear trouble. A bulging black drum on incision gave vent to a quantity of bloody fluid. The following day there was a chill with fever leukocytosis. As the chill was repeated, it was decided to investigate the lateral sinus. A normal mastoid process was found with a tear in the sinus wall, although the latter could not be found. The sinus was thrombosed; the broken down clot and the sinus wall were removed and gauze drains were inserted. The internal jugular vein was not disturbed, as the phlebitis had already extended beyond the point of resection. Shock following the operation was profound, but after twenty-four hours the patient rallied, making a complete recovery with a dry normal ear. The author believes that incision of the ear drum would have improved the drainage from the middle ear where the clot had become infected, and that the infection would not then have spread to the sinuses.

HUNTER, Philadelphia

A PECULIAR REFLEX PHENOMENON. P. M. VAN WULFFTEN PALTHE, Brain **48**:476 (Dec.) 1925.

By stimulating the upper lip immediately below the nasal septum, or the mucous membrane of the latter by a current of air or ether spray, a peculiar reflex phenomenon is elicited which consists in: arrest of breathing in any phase; movement of the floor of the mouth and of the root of the tongue; movement of the larynx; increase of pressure in the nasal cavities. This reflex resembles in many respects the swallowing reflex except that in the latter the swallowing act always occurs at the end of expiration and the intranasal pressure is not as high. The centripetal part of the reflex path is probably formed by the second branch, the gasserian ganglion and the root of the trigeminal nerve; a few clinical cases are reported in which injury to this branch destroyed the reflex. The author thinks that biologically this reflex may be considered as a protection against the entrance of harmful substances into the larynx and pharynx, and that it likewise acts during unconsciousness. The unpleasantness which arises when a strong current of air strikes the face is explained by this phenomenon. It is suggested that this reflex might likewise have localizational value in distinguishing between disorders in trigeminal sensibility of peripheral origin and those due to more centrally situated lesions, above the linking center of the cerebral nerves.

STACK, Milwaukee.

MICROSCOPIC CONDITION OF SPINAL CORD IN PEMPHIGUS. I. L. MCGLASSON and W. KEILLER, Arch. Dermat. & Syph. **12**:829 (Dec.) 1925.

Lesions of the spinal cord in pemphigus are not constant. In the case reported in detail the most striking feature was the enormous number of corpora amylacea. These were especially marked in the posterior columns, in the subpial neuroglia and in the glial septums which stretched inward from the surface, as well as in the posterolateral columns and around the central canal. Capillary hemorrhages were the second most striking feature, especially in sections of the lower thoracic cord. The apparent disappearance of many of the anterior horn cells and cells of the nucleus dorsalis and marked degeneration of those that remained was the third most striking feature. The lesions found in the cord are believed to be the result of profound toxemia and in no way accountable for the skin lesions. In a second case, to be reported in more detail later, Keiller found no corpora amylacea, but he did find degeneration of the nerve cells and occasional microscopic hemorrhages. The first case was that of an exceedingly fat woman; in the second case the patient was extremely emaciated. Keiller questions whether possibly the extreme emaciation and presumably carbohydrate hunger might account for the absence of corpora amylacea.

SCHUMACHER, Philadelphia.

A METHOD OF PALPATING THE LOBES OF THE THYROID. FRANK LAHEY, J. A. M. A. **86**:813 (March 20) 1926.

The author describes a method of palpating the thyroid gland, which furnishes information regarding the lateral lobes and posterior surface so often missed because the usual method gives information only regarding the anterior surface. The chin is elevated and rotated slightly toward the side to be palpated. The original portion of Dr. Lahey's method is placing the ball of the thumb against the lower lateral portion of the thyroid cartilage and the upper ring or two of the trachea and then dislocating the trachea laterally, avoiding choking. The fingers of the other hand now press deeply inward behind the sternomastoid muscle and the thumb is brought anterior to the muscle; the dislocated lobe is then palpated between the thumb and fingers. The method is most valuable in cases of slight or doubtful enlargement. It is also valuable in determining whether or not local enlargements are adenomatous or malignant.

CHAMBERS, Syracuse, N. Y.

THE OPERATIVE TREATMENT OF GASTRIC CRISES. MCDONALD CRITCHLEY and J. M. WOLFSOHN, J. Neurol. & Psychopath. **5**:318-331 (Feb.) 1925.

The authors comment on the various forms of operative procedure attempted in this type of case and report five cases treated by ligation or section of the posterior nerve roots. Two patients died from three to four weeks after the operation. In the three patients surviving, only one showed definite improvement and this has been maintained for three years. Another patient remained free from pain and vomiting for twelve months. The other patient showed improvement for three months only. Two cases are reported with treatment by both anterior and posterior root division. In both cases no improvement occurred. The authors theorize in the light of other reported cases on the nature of gastric crises and plead for a more thorough and systematic investigation into the nature of tabetic crises. The bibliography is unusually complete and contains sixty-five titles.

POTTER, Akron, O.

PSYCHIATRIC PHASES IN VOCATIONAL GUIDANCE. HARRY M. TIEBOUT, Ment. Hyg. **10**:102 (Jan.) 1926.

Vocational guidance must take into account not only the intellectual capacities of the person but also his emotional or affective life. In this paper only one phase of the affective life — the inferiority complex — is discussed. Inferiority may be met in one of several ways. It may be squarely faced and successfully compensated. The person possessed of such feelings may overcompensate, or he may succumb to his inferiorities. In vocational guidance cognizance must be taken of these feelings of inadequacy or of inferiority whenever present. The need of compensating for them must be kept in mind in the selection of the job or vocation. However, these job compensations need not be specific, but may be part of a general adult adjustment. Vocational guidance should concern itself with the entire personality adjustment of the individual.

SCHUMACHER, Philadelphia.

FASTING AS A CAUSE OF CONVULSIONS. HUGH JOSEPHS, Am. J. Dis. Child. **31**: 169 (Feb.) 1926.

The author noted in a number of young children that convulsions tended to occur in the early morning, before breakfast, or at night only after the child had missed his supper; and after a meal only when the child had vomited that meal. Blood studies indicated that in this group the convulsions were associated with a lowered blood sugar concentration. The tendency was toward spontaneous recovery from the attack; when this was delayed, administration of glucose produced rapid recovery. Cases of convulsions due to organic brain lesions and cases of idiopathic epilepsy were excluded from the study.

VONDERAHE, Cincinnati.

EXPERIMENTAL STUDIES ON IMMUNITY IN HERPES SIMPLEX. S. S. GREENBAUM and M. J. HARKINS, Arch. Dermat. & Syph. **11**:789 (June) 1925.

Experiments were conducted on rabbits to ascertain the possibility of inducing immunity to herpes simplex. Three strains were used, of varying virulence, in subcutaneous, corneal and intracerebral injections. The results seem to indicate that some degree of local immunity is secured, this being greater with the more potent strains. It is possible that some slight antibody production occurs, but as complement fixation tests were uniformly negative, these are evidently not of the amboceptor type.

Some interesting experiments on filtration lead to suspensions in glycerol passing the Mandler filter, while those in glucose hormone broth fail to do so.

A good summary of recent experimental work on herpes is included.

ANDERSON, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 21, 1926

SANGER BROWN, M.D., *President, in the Chair*

PSYCHOSIS IN A GIRL AGED 6 YEARS. DR. BERT I. BEVERLY.

Clinical History.—This white, English-American girl was admitted to the Children's Memorial Hospital in August, 1924, with a history of having been well until three weeks previously, when she became extremely unstable emotionally, very fearful of water, animals, etc.; complained of seeing men trying to get her, and was afraid of animals — cats, dogs, etc. She cried bitterly without apparent provocation; laughed uncontrollably for from twenty to thirty minutes at a time "at the doorknob"; fought and used extreme profanity at any one approaching, and displayed all possible moods. Her condition became rapidly worse.

The developmental history was normal. The only illnesses were pertussis and measles at 4 years, and an occasional cold. There were no recent febrile attacks of any kind. The patient is the third of four children. An older sister has some evidence of a glandular disturbance. A younger brother is having difficulty in school. All are well. The father is well and apparently normal. The mother has always been "very nervous." She has a quick temper and frequently has periods of depression for several days at a time. She worries a great deal and often has subjective vasomotor disturbances, choking sensations and other somatic complaints. A maternal aunt has outbreaks when she cries for hours, throws objects, is unable to sleep or work for days and becomes unmanageable. The maternal grandmother is described as "highstrung," having a violent temper, and worrying a great deal. A maternal greatgrandmother had several attacks of paralysis from nervous shocks, such as the death of her daughter.

Examination.—On admission to the hospital the child was rather pale and poorly nourished, and appeared moderately ill. Her behavior was essentially as described above. She appeared confused, yet was very alert at all times: was hyperactive and fearful, slept little at night and at times was noisy and destructive. She screamed, fought, cried, laughed, complained of seeing animals and refused to talk after the first two days in the hospital. After refusing food for two days she was tube-fed once and always ate poorly.

There was no fever. The physical, including neurologic, examination gave negative results. The superficial reflexes were normal; there were no areas of hypoesthesia or hyperesthesia. The blood examination, including white and red count, differential count, hemoglobin and Wassermann test gave negative findings. The blood chemistry was normal. The eyegrounds were normal. Roentgenograms of the teeth, skull and sinuses of the head gave no evidence of disease. The tonsils had been removed. Spinal puncture was negative. The pulse rate was between 75 and 85. These findings have not changed at any time.

During the first two days in the hospital the girl talked freely and coherently at times, and was sufficiently cooperative to give a fairly satisfactory psychologic examination. By psychometric tests (Stanford-Binet) she was found to have a mental age of about 6 years and 10 months. Her chronologic age was 5 years and 7 months. Although silly at times, her responses were usually logical if the tests were repeated a sufficient number of times. Her attention was difficult to control. Mentally she was alert and oriented. She jumped from one subject to another; laughed one minute, and cried bitterly the next. After the first few weeks in the hospital her activity and fearfulness decreased. For several weeks she was content to lie in one position for many hours at a time. After a few months she began to say a few words at times when urged and displayed slight interest in her surroundings. Her talk and actions were always very bizarre, however, and she never played with other children of the ward for more than a minute or two at a time. Incontinence and enuresis were present the greater part of the time. She was discharged from the hospital in December, 1924.

Course.—Since discharge, the child's condition has improved. She still volunteers little information, however. She never plays with the other children in the family, shows little interest, and is at times unmanageable. Her statements and actions are always peculiar. She often stops whatever she is doing, cries, wrings her hands and appears frightened.

Many important factors are manifest in the personality history. The patient is described as having been always nervous. She was always good and loving, the pet of the family, relatives and neighborhood. She was never punished, always obeyed directions and "never did anything wrong." She was always fearful and cried easily. At one time she nearly fainted when a brother fell and suffered a minor injury of the head. She was always a follower and "gave in" to other children without the slightest resistance. Parents and relatives considered this child "different" from the other members of the family. The fearfulness and instability became more noticeable about a year previous to the onset of the acute condition. She gradually became frightened more easily, cried more easily, was more easily disturbed, was more hyperactive and restless, and slept poorly. The acute condition, then, is apparently an exaggeration of personality traits which have been present since late infancy at least.

DISCUSSION

DR. PETER BASSOE: Dr. Roberg referred this child to me July 24, 1924. She then appeared timid and frightened, but was not actively disorderly. Her parents said she was afraid of "beggar-men" and of rats. I could find nothing abnormal on neurologic examination.

DR. RALPH C. HAMILL: It was interesting that the patient showed the period of acute confusion just after coming into the hospital; it became more and more marked while she remained in the hospital and in that way resembled an adult psychosis. She showed no physical signs, but at times was almost catatonic. I have no idea what the condition is.

GUNSHOT INJURIES OF THE BRACHIAL PLEXUS. DR. LEWIS J. POLLOCK.

Gunshot injuries of the brachial plexus in the World War occurred in about 7.7 per cent of peripheral nerve lesions. These injuries do not lend themselves to the simple classifications found in civil practice. This was due not only to the varying planes of injury by missiles, secondary changes of

hemorrhage, infection and fibrosis, but also to the frequent variations in the formation of the brachial plexus. Anatomically the cases could be divided into supraclavicular and infraclavicular lesions, and into upper, middle and lower brachial plexus lesions. None of the cases accurately corresponded to any classification, and it was found useful to use a diagram such as has been described by Meige to indicate the probable location of the injury. Intravertebral root lesions occurred only when evidence of spinal cord injury existed. Paralysis of the latissimus dorsi and pectoral muscles occurred more frequently when the primary cords were injured. The supinators frequently escaped in a lesion corresponding to the fifth cervical root. Lesions of completely formed nerves and of roots produced complete paralysis, whereas other lesions produced incomplete paralysis. In middle plexus lesions, when the median nerve was slightly injured, the opponens pollicis seemed to be the most vulnerable muscle. The sensory loss was rarely coextensive with the motor disability. It was not as reliable an index to the extent or severity of the injury as was motor loss. At times complete sensory loss was found when motor disability was slight. This was particularly true of lesions of the inner cord. Sensory loss coextensive with motor paralysis was seen usually in lesions of the roots. The sensory area of the outer secondary cord of the brachial plexus was found to be represented by the sensory distribution of the musculocutaneous nerve and that part of the median supplying the radial side of the palm, the thumb and part of the index finger. This part of the median sensory supply would then correspond to the anterior division of the seventh cervical root. Seventy-nine cases were studied. Ten of these were found to be associated with a spinal cord lesion producing a Brown-Sequard paralysis. There is a possibility that the observation that the fibers for pain cross more directly in the dorsal region than in the cervical may in part be a misinterpretation, inasmuch as some of the sensory loss on the side of the lesion was the result of injury to roots and plexus in the cervical region.

Brain Changes in General Paralysis Treated with Tryparsamide. Dr. Peter Bassoe and Dr. G. B. Hassin.

This article appears in full in this issue of the Archives.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 22, 1926

S. F. Gilpin, M.D., *President, in the Chair*

Neuro-Recurrence: Bilateral Facial Palsy. Drs. C. S. Potts and S. S. Greenbaum.

A negro, aged 29, was first seen in the skin clinic June 25, 1925, when he was treated for a chancre of the right forehead of two weeks' duration. The dark field was positive, the Kolmer complement fixation test was negative, but the Kahn precipitation test was strongly positive. The general physical examination was negative. Summary of treatment: June 25 and 29, bismuth; July 2, 9 and 16, neo-arsphenamine 0.45 Gm.; September 24, neo-arsphenamine 0.9 Gm.; October 5 and November 2, neo-arsphenamine 0.45 Gm. The patient had severe reactions after all neo-arsphenamine injections.

He then disappeared from the skin clinic, and on Dec. 21, 1925, presented himself in the neurologic clinic with a complete right facial palsy of the peripheral type. One week later he again reported and was found to have complete double facial palsy. He had been having severe headache for several weeks. The examination of the spinal fluid revealed: 161 cells per cubic millimeter; no increase in globulin; colloidal gold curve 1111000000; weakly positive Kolmer test. The eye examination gave negative findings, and, while hearing was apparently normal, low tones were not heard at quite a normal distance.

DISCUSSION

DR. JAMES H. LLOYD: This is apparently a case of early neurosyphilis following insufficient treatment with neo-arsphenamine. The man had a chancre in June, 1925, and in December he had developed a double facial paralysis, due evidently to cerebral syphilis. Dr. Nonne has recently stated his belief that since the use of arsphenamine, there has been an increase of early neurosyphilis following insufficient treatment with that drug. Gennerich has attempted to explain this by saying that the early treatment with arsphenamine interferes with the florid or secondary stage. This is frequently seen in cases of tabes and general paralysis, and it may occur also in cases of cerebrospinal syphilis. If the original treatment is insufficient, it interferes with the secondary reaction, and leaves the patient in worse condition and exposed to neurosyphilis. This secondary reaction is evidence that nature is trying to confer immunity. Syphilitic patients would possibly be better off if they were allowed to have a full and frank secondary eruption. I know that I have seen many more of these cases of early neurosyphilis since the introduction of arsphenamine than I ever saw before.

Dr. Klauder, in a recent paper, makes the statement that bismuth does not interfere to the same extent as arsphenamine with this immunizing reaction. We shall see.

DR. T. H. WEISENBURG: How does Dr. Lloyd explain the absence of the usual secondary manifestations of syphilis in most cases of tabes and general paralysis in view of his statement that these diseases develop because of the absence of secondary reactions as a result of early arsphenamine treatment?

DR. JAMES H. LLOYD: I do not explain it. I only know that many cases of tabes and general paralysis have no history of a secondary reaction. Possibly this is due to insufficient early treatment interfering with the florid stage.

DR. C. A. PATTEN: What is the shortest space of time that has elapsed between the initial lesion and the cranial nerve symptoms? I ask because during military service we had a man admitted in June from overseas who gave a history of having had an initial lesion in April, and had developed a facial paralysis on one side and, two weeks later, on the opposite. There was another man on the neurosyphilis service, who had developed cranial nerve symptoms three months after the initial lesion. I do not know how much reliance we can place on the history, but I would like to know the usual length of time between the initial lesion and the onset of the cranial nerve symptoms.

DR. S. GREENBAUM: Neurorecurrences are syphilitic manifestations and not toxic reactions due to the arsenical used. The fault is with the patient, not with the drug.

DR. S. F. GILPIN: The point that interests me is whether the patients do as well if treated as soon as the chancre is diagnosed, as this patient was treated, or whether they eventually do better if not treated until the disease

is further developed. This question is not new. At the meeting of the American Medical Association in 1914, the chairman of the Section on Mental and Nervous Diseases prophesied that in ten years we would reap a crop of neurosyphilis because we depended too much on drugs other than mercury. It is not unusual to see a patient treated intensively with arsenicals for from six months to one year from the chancre stage and then develop severe neurosyphilis. It would seem that the spirochete becomes immune to one drug and that the change to another drug produces better results. At least, the cases doing badly on arsenicals seem to respond well to mercury and iodides.

Dr. Charles S. Potts: I cannot answer Dr. Patten's question dogmatically. I know, as all do, that neurosyphilis may appear very early. Many years ago, I saw a man who developed syphilis of the spinal cord three months after the chancre appeared. I think such early cases are not frequent, but they are probably more frequent now than years ago. There is surely more attention directed to them. I believe in the use of mercury and iodine in the treatment of neurosyphilis. Many patients were cured by the use of these drugs before the days of arsenicals. This is especially true in early cases; arsphenamine may be preferable later.

Report of a Case of Epilepsy with Convulsive Movements Limited to the Upper Half of the Body. Dr. Raymond Waggoner.

This case is presented because of several unusual and interesting features. The patient is a boy, aged 8½, born of American parents, who first came to the Infirmary for Nervous Diseases in 1922, complaining of "night terrors." His father, aged 31 years, is living and well; his mother, aged 29, is at present eight and one-half months pregnant. She has had four other pregnancies. The first ended in a miscarriage at two and one-half months; during the second, that of the patient, she menstruated regularly until the seventh month — it was otherwise normal; the third ended in a miscarriage; the fourth was normal; the fifth, the present one, has been apparently normal. A sister of the patient's father was treated at the clinic twelve years before for chorea and at present has active pulmonary tuberculosis.

Birth and developmental history to the age of 4 years was normal. At the age of 4, the patient had a rather severe fall, but was not unconscious at any time; shortly afterward he began to stammer. This condition grew progressively worse. Three months later, after seeing a motion picture which showed many crocodiles and snakes, he awoke during the night screaming and talking about snakes. After this nightmare, the child became frightened easily and was afraid to stay alone. His appetite became poor and he did not sleep well. At the first physical examination he showed only a peculiar facial expression, widely spaced teeth and a moderately high-arched palate. He was given a mild sedative and his mother was instructed concerning his diet. He came back one week later apparently much improved.

Six months later, the mother noticed that he would drop his head on his chest for a moment, then throw it up and roll his eyes. These spells became progressively more severe and more frequent, but never lasted more than a few seconds.

In March, 1925, he was taken to Jefferson Hospital for one week for observation. The findings there were essentially negative except for a low hemoglobin and red cell count and a white cell count of 32,000. For the nine months following he had no attacks and was much more alert. During this time he was taking phenobarbital.

December 7, the patient reported back to the Infirmary complaining of momentary spells of unconsciousness with convulsive movements of the head and arms, but with the convulsive movements in no way affecting the lower part of the body. These had developed during the month before, and were becoming more frequent; sometimes there were as many as 100 in one day. They were said to be more frequent when he attempted to think, or during an emotional strain of any sort.

The attack consists of a dropping forward of the head and a waving of the arms, followed by ten or twelve clonic movements of the head and arms. The lower half of the body is absolutely free from muscular change. The patient does not drop whatever he may have in his hands, does not fall and, as a rule, does not know that anything has happened. He pauses for the moment in whatever he may be doing and then proceeds from the point at which he stopped. He is usually tired and drowsy, especially after several attacks.

Examination at this time reveals one or two additional points. The patient now has a slight tachycardia with a pronounced systolic murmur at the apex. The eyes are normal. Roentgenograms of the skull show only a possible slight enlargement of the sella. Dental roentgenograms are negative except for wide spaced teeth. Urine, blood Wassermann and stool examinations gave negative results. Muscle reactions show practically no change in the threshold to electrical stimulus. Blood calcium content is at a high normal level. Since the child has been in the hospital he has had a varying number of attacks—from one to twenty-seven in one day. He has had phenobarbital ½ grain (0.03 Gm.) at night since he has been in the hospital and is apparently improving.

The essential points in this case are the mother's menstruation during pregnancy; the child's stammering; the night terrors; the development of periods of momentary unconsciousness without convulsions about six months later; the high leukocyte count at the time of his examination at Jefferson Hospital; cessation of attacks for nine months; then again development of these attacks accompanied by convulsions. The patient has certain definite stigmas, and—most interesting of all—the bilateral involvement of the head and arms with absolutely no involvement of the lower part of the body at any time. The patient developed heart disease at some time during the interval between his first and second visits to the dispensary.

In reviewing the literature, I have not found a single case report of epilepsy with bilateral convulsions limited entirely to the upper half of the body. Gowers mentions that epilepsy may manifest itself in this form but cites no specific case. Epilepsy is often associated with night terrors, and Block states that epileptic attacks may be represented by night terrors.

The etiology in this case is not clear; no definite organic basis could be found. It may be due to fright or to an emotional upset of some sort. Starr, in his table of the causes of epilepsy, places fright in the second place. Gowers states that in his series of cases in which a cause could be found, one-third were due to emotional disturbances of some sort. In my case the attacks are much more frequent when the patient is under emotional strain or if he is asked to think.

DISCUSSION

DR. C. W. BURR: As Dr. Waggoner has already said, the most interesting thing in this case is that the convulsive movements involve the head, neck and arms, with absolutely no convulsive movements of any kind in the legs. Several times in the clinic, while standing, he had attacks without any involvement of the legs. The boy is momentarily unconscious in the attacks. Mental

effort seems to precipitate them; for example, when a question is asked requiring thought to answer, an attack usually follows.

Dr. Alfred Gordon: Convulsive movements of the body—neck, arms and head, and none of the legs—and going to sleep or being dazed for some time after an attack, I think, eliminate the possibility of petit mal, even if the patient does not fall. I have seen cases of this sort, cases in which the mother gives a history of miscarriages and the child develops epilepsy, which might be considered as a manifestation of hereditary syphilis.

Dr. James H. Lloyd: It is possible to explain this case as an example of epileptic automatism. I have heard of one patient who ran along the street before falling in the fit, and of another who partially undressed himself, but I must confess that I have never known anything exactly like this case of Dr. Burr's. It seems like a double or bilateral jacksonian fit.

Dr. Raymond Waggoner: I believe that the patient is unconscious, because we can elicit no response while he is having an attack. He gives no reaction whatever to external stimuli.

The Relation of Trauma to Neuropsychiatric Conditions as Observed in a Compensation Court. Dr. S. F. Gilpin.

In the routine work of a physician dealing with mental and nervous conditions, both in hospital and private practice, the friends and relatives of the patient frequently call attention to trauma, recent or remote, as a factor in the causation of the particular condition from which the patient is suffering. After obtaining a complete history and carefully weighing the evidence, he usually decides that the patient is suffering from disease due to a specific organism, to some congenital defect or to some hereditary taint, each sufficient in itself to produce the disease condition. Unless he finds gross and unmistakable evidence of injury directly connected with the symptoms, which to his mind are clearly related to the trauma, he is apt to consider the injury merely an incident in the life history of a patient previously predisposed and practically certain to develop the disease from which he is now suffering. However, when we come to study pathology in the light of compensation claims, we find trauma given as the cause of nervous and mental disease ranging all the way from general paralysis to hysteria. It is my purpose to discuss this from the standpoint of actual cases and not from theoretic reasoning.

It is true that syphilis may develop after a slight trauma, though not from trauma caused by an industrial occupation. We know that it is the result of a specific infection with the *Treponema pallidum* and that the diseases of the nervous system which it produces are called tabes, general paralysis and cerebrospinal syphilis. This fact is so well recognized that no one, even in court, has the audacity to claim that these diseases are the result of trauma, but that they are brought on earlier in the career of the claimant and that the symptoms are aggravated by trauma.

A man while at work received a severe injury resulting in fracture of the pelvis. From this he recovered as completely and as rapidly as any person ever recovered. While in the hospital it was noticed that he was slow mentally. Examination on his discharge from the hospital showed well marked physical signs of general paralysis. The diagnosis was confirmed by positive laboratory findings. A claim was brought and allowed for full compensation, the patient being completely disabled mentally from general paralysis, when considered for the requirements necessary to a man doing a laborer's work. He entered a state hospital, his disease ran the regular course and he died of

the disease in four or five years. There is no medical evidence in this case that death was hastened by the severe trauma.

A common sense medical point of view would suggest that a person with general paralysis suffering a trauma, should be compensated for the time lost by reason of his injury but that industry should not be required to care for a disease we might well call self-inflicted. Because of the intense light produced by the shortcircuiting of an electric current, a workman stated that he was made partially blind. A claim for compensation was made. Examination showed that the patient was suffering from early tabes. It is not known that intense flashes of light produce optic atrophy. We all know that this is frequently the most hopeless symptom of tabes. This patient had not noticed the gradual loss of vision until the disturbance temporarily produced by the flash of light brought it to his attention. A workman fell some distance onto a pile of iron and steel rubbish. He was seriously injured but made a good recovery. Some months later he noticed failing vision. Examination showed optic neuritis with developing atrophy. He presented physical signs and laboratory evidence of syphilis of the central nervous system in all probability of the interstitial type. A claim was made that even if the blindness was due to syphilis it had been developed or aggravated by the injury sustained in falling. Trauma certainly is not a cure for syphilis of the nervous system; but how many patients with cerebrospinal syphilis suffer with optic neuritis followed by atrophy and blindness in which there is no history of trauma, and what physicians would consider trauma a factor in such a case unless compensation were involved?

In severe trauma to the head, as in fracture of the skull, it is not uncommon for epileptiform attacks to develop. Usually these attacks come on within a year after the trauma. The attacks are similar to those of essential epilepsy, and operations on the skull usually are disappointing in that no local lesion is found and the patient is not relieved. Such patients should be entitled to full compensation, but why should we physicians not admit that a patient may have a fractured skull and make a complete recovery, not befogging the issue by stating all the dire things that may happen?

As we study the history of the usual case of tumor of the brain or spinal cord, seldom do we believe trauma to be a cause, yet if a patient has a trauma while working and later develops a tumor, some one is willing to say the injury was the direct and exciting cause. A man received a head injury which did not produce unconsciousness nor fracture of the skull. A day or two after the accident he complained of headache and vomiting. Later he entered Jefferson Hospital where a diagnosis of cerebellar tumor was made. This diagnosis was confirmed afterward by necropsy at another hospital. The tumor was shown to be a glioma. Is there any evidence that a tumor such as this could develop and give marked symptoms in two days? Rather, was not the tumor developing and the trauma only an incident in the life of that man? Such an opinion was given in the case. Yet we were asked whether there was no possibility of trauma causing the tumor. We could answer only that many things may be possible which are not probable.

A young man was thrown violently on his head and shoulders striking the earth with sufficient force to fracture his jaw. In a few months he began to develop atrophy of the muscles of his upper extremities. He was shown before this society as a patient with syringomyelia because of sensory symptoms. Since then the atrophy of muscles has progressed until his arms are hanging helpless and he has complete dissociation of sensation in his arms and shoulders. He has also increased reflexes in the lower extremities. Are

we right in this case in assuming that trauma has been a factor in developing a cord lesion? It may be only a coincidence but there is the proper time relation between the trauma and the beginning of symptoms. Some writers think syringomyelia may develop because of trauma, though positive proof seems impossible.

Claims are sometimes made in other mental conditions than general paralysis. A patient with mania may receive an injury because of his motor activity combined with lack of attention to his surroundings.

It is rather becoming the fashion, instead of recognizing fifty-seven varieties of dementia praecox, to deny there is such a disease. However this may be, most physicians with practical experience realize that there is a troublesome affliction which is associated with adolescence. Because a patient during the confusion associated with dementia praecox receives an injury, an effort is made to establish the claim that the injury is the cause of the mental condition. Outside of a compensation court, whoever heard of trauma causing dementia praecox?

One of the frequent claimants of compensation is the hysterical person. These patients complain so loudly and present such marked symptoms that it is almost impossible to convince a referee that work is the best remedy for their cure. The patient has usually received an injury of a trifling character. It may be a muscle strain or a light blow. He cannot work because of the intense suffering which he noisily insists has been present only since his accident. He is so constituted that the $12 to $15 a week he receives as compensation makes him happy. One such patient was found to be working in his garden with no pain in his back if he thought he was not observed. One was examined and reexamined over a period of four years, and all of the doctors concerned came to the conclusion that he needed a neurologist. Examination showed a typical hysterical hemianesthesia. The patient at last became tired fighting for compensation and went to work for double the amount.

It may be well for us to ask ourselves, as neurologists, where the rapid multiplication of compensation claims is leading. We may say that it is well for industry to take care of all such patients and relieve the taxpayer of caring for them in a public hospital. We may forget that industry passes the bill on to the ultimate consumer, the payer of all bills, taxes included. We should remember also that a dollar taken without value given means that some one is a loser and some one is a robber. We realize that compensation is awarded by laymen, men who do not understand human ills and injuries, and who are befogged by disagreeing doctors. When a layman, possibly a politician, with his ear to the ground and his eye on the voter, listens to the opposing statements of physicians, how can we expect him to analyze the testimony and know who is correct, and how can we blame him if he favors the poor workingman at the expense of the public? What is right about the rule of allowing full compensation because it is established that trauma aggravates neurosyphilis in its various forms, or hastens the development of a glioma, or brings to a focus a dormant case of dementia praecox? Trauma is but an incident in the course of these diseases, and the patient should be paid compensation for the trauma; and a very careful adjustment should be the rule when considering the aggravation of preexisting disease by trauma. Hysteria should be recognized as due not to trauma, but to the suggestion of compensation for trauma, and should not be compensable and thus be banished from the list of diseases in any way associated with trauma. Such a condition of affairs will not be until the human race improves and competent medical boards are called on to adjust compensation.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 2, 1926

I. Abrahamson, M.D., *President, in the Chair*

A Case of Neuroretinitis with Recovery. Dr. Irving J. Sands.

J. W., a man, aged 32, married, white, on Oct. 7, 1924, complained of increasing loss of vision for one year. The family history was unimportant. He was born in Ireland, and passed through a normal infancy and childhood. He had two years of schooling. He emigrated to the United States in 1916. He denied venereal disease, the use of drugs and excessive use of alcohol. His wife was never pregnant. On Oct. 1, 1918, he received a gunshot wound in the left wrist, and was in an army base hospital two months. He worked on a farm until the latter part of October, 1923, when he stopped work because of dimness of vision.

Physically he was well developed, but undernourished. The heart and lungs gave negative findings; the systolic blood pressure was 135, diastolic 75. Gait and station, coordination, and reflexes were normal; there were no sensory disturbances. The pupils were equal and regular, but reacted sluggishly to light and in accommodation. There was marked blurring of both disks, especially on the nasal borders; the retina at the edge of the disks was also involved; the disk vessels were congested, but no hemorrhagic areas were seen. Vision was 10/20 in each eye. There was no other cranial nerve lesion. Urinalysis was negative. The blood count showed 4,350,000 red cells, 6,800 white cells; the differential count was: polymorphonuclear leukocytes 63 per cent; lymphocytes, 35 per cent; eosinophils, 2 per cent. Blood Wassermann reaction was negative. The spinal fluid, of normal pressure, showed three cells, globulin one plus, sugar present, negative Wassermann reaction, and colloidal gold curve 0000111100.

Dental examination revealed periapical abscess of the upper right first bicuspid, and an infected root of the left upper first bicuspid. Roentgen-ray examination of the skull sinuses showed multilocular, symmetrical and normally illuminated frontal sinuses, normal ethmoids and normal maxillary antrums. Nose and throat examination revealed large, infected tonsils and hypertrophied right turbinates.

The diagnosis made was: neuroretinitis, bilateral. Dental care was immediately instituted.

On Jan. 30, 1925, there was both subjective and objective improvement in the eye condition. This continued. Tonsillectomy was done October 5. On Jan. 22, 1926, eye examination revealed normal disks, vessels normal, vision 20/20 in each eye.

DISCUSSION

Dr. G. H. Hyslop: Did the patient have any central scotomas?

Dr. Sands: No.

A Psychanalytic Introduction to a Study of the Conduct Disorders of Lethargic Encephalitis (with Outline of Plan for Psychosocial Reconstruction). Dr. L. Pierce Clark.

An hypotheses on which a theory of the conduct disorders of postencephalitic patients may be based is as follows: "The nature of the diffused lesion in the infectious process leaves residual lesions in the brain, which I designate as the ego organ of the mind, and because of this lesion the free

flow of libido imprisoned in the lesion is incapable of healing and developing the proper integrative function of the person. This disturbance of the imprisoned narcisistic libido periodically manifests itself in various sorts of conduct disorders at the different levels of the wounding of the ego libido. Under various types of adaptations the individuals regress to different instinctual levels, thus giving a variety of conduct defects proportional to the degree and extent of the lesions. I consider the nature of the disorder as a pathoneurosis, organically conditioned, in which there is insufficient power of normal integration of function of the ego organ of the brain." Dr. Clark then briefly indicated the extent of the social problem and the types of insufficient recoveries and relapses in a series of cases, and detailed the psychanalytic reeducation in one case over a period of ten months; and finally cited in detail the beginning analysis of a very severe case of conduct disorders in a postencephalitic patient affected with the parkinsonian syndrome. The results of analytic sessions are by no means final, as the length of time has been insufficient to show what effect psychanalysis might have in one suffering from a degenerative brain lesion. However, it is just possible that through analysis the symptoms may disappear for a time and give an opportunity to build up the psyche to keep abreast of the degenerative process. Dr. Clark then outlined a plan, with estimated cost, for organizing an observation home for the intensive study and training of a group of cases along psychanalytic principles.

DISCUSSION

Dr. M. W. Raynor: I am much interested in Dr. Clark's paper, as I have at Kings Park about fifty children, most of them approximately 12 years of age. We have had about eighty patients under 16 in the last two years. All have been certified as having psychoses. There has been considerable deviation in their conduct, and some of them have shown hallucinations and delusions. We have two cottages, one for the boys and one for the girls, each holding twenty-five patients. We chose the personnel, nurses, an occupational therapist and a school teacher, with special reference to the care of these children. The children presented a practical problem; it was impossible to care for them in wards with adults, and it was desired to provide a proper psychologic atmosphere. They have been trained to care for themselves and their cottages. Recreation has been organized and carefully supervised. They have been given a certain amount of academic instruction, to which they have responded very well. The whole point has been to stimulate and maintain an interest in reality. Much improvement has taken place in the majority, but we have been unable fully to meet the situation because the personnel has not been sufficient. We have not been able to undertake detailed mental analysis, but we have made a careful study of the personalities and the new psychologic problems which have presented themselves to these children. When the children reach the age of about 12, a new problem arises. They begin to show a quite definite sex trend; the sex abnormalities seem much more prominent and become a more serious problem.

Improvement is not rapid, but it is seen in general conduct and in emotional reaction; the explosive episodes are not so frequent nor so marked. Many of these patients showed some spasticities, not localized but general, which slowly cleared up. A number had the typical breathing syndrome, which has been about the last feature to show improvement; but in some cases this also improved markedly. I believe that it is necessary to analyze the mental condition if we are to get a further marked improvement.

MR. T. E. UNIKER (by invitation): For several years I have had under observation a number of postencephalitic patients in a group organized by Dr. Clark. I thoroughly agree with Dr. Clark's and Dr. Raynor's conclusions. It is obvious that the immediate problem is not only to create the proper home atmosphere where the different types of occupational interest can be employed, but also to provide some therapy that will take care of the compulsive, sadistic and masochistic behavior reactions which are so marked in these cases. Psychanalysis offers a means by which we can reach these patients, especially through the fantasy method described by Dr. Clark. Under this method, or a modified analysis, all our cases showed immediate improvement. The respiratory and parkinsonian syndromes, together with the conduct disorders, have all abated. It would seem that the plan suggested by Dr. Clark for a small number of cases carefully studied and analyzed would provide a means for organizing therapy for larger groups.

DR. L. GRIMBERG: I am not convinced that the majority of postencephalitic disorders are really due to encephalitis. In postencephalitic disorders are we dealing with narcissism? I have watched a number of children, and have seen no real improvement. There has only been a change from one condition to another. We notice a similar change in plain parkinsonian disease. To my mind also there is absolutely no relation between the severity of the lesion of the brain and the amount of conduct disorder. I have seen patients with very slight attacks of encephalitis who showed great conduct disorder, and I have seen patients with severe atfacks show very slight disorder. I cannot believe that the parkinsonian tremor is due to a masturbatory tendency in the patient. I think it is due to an organic lesion of the extrapyramidal tracts. I am not a psychanalyst, but it seems to me that a number of patients discharged from Kings Park have not shown much improvement, whether psychanalyzed or not.

DR. SMITH ELY JELLIFFE: Dr. Clark says there is no correlation between the cerebral lesion and the symptoms. That is not so. A great many sound correlations between the symptoms and the lesions have been made by a number of observers. Any one who has gone over Kleist's many studies knows that there are many such correlations, and he is but one of the many observers—Zingerle, Förster, Naville, Hanard, Winner, Hohmann, et al. The symptoms do not necessarily have to be interpreted in a positive sense. They may be interpreted in a negative sense, and when I use these two words, I am reminding you of Hughlings Jackson's idea about the "dissolution of function," that a lesion in a certain place does not necessarily produce symptoms from that place, but possibly from some other place; in v. Monakow's diaschisis discussion, there is an elaboration of Hughlings Jackson's conception. Furthermore, I cannot agree with Dr. Clark when he says that there could be no bridge between the body and the psyche, nor am I certain that he quotes Sherrington correctly. In the first place, there was never any separation. There is no need to build a bridge, because there was never any gap. Soma and psyche are one; always have existed together, and always will. They represent in psychic or bodily manifestations different phases of the evolution of the functioning of a human being, and different modes of reaching what the human being wants to reach. In disease processes, therefore, different grades of dissolution of function may occur, according to the position of the lesion. In that sense I am a somaticist. I can refer Dr. Clark further to Dr. Küpper's studies on the so-called location of psychic functions; to Wilckins' paper in which are discussed three cases. His second case is of great interest: an

encephalitis occurring in a feebleminded boy who also showed a dementia praecox reaction. Wilckins has given a beautiful description of the respective localizations of the processes which produced not only his feeblemindedness and dementia praecox, but also the symptoms that newly arose in the course of the encephalitis.

Dr. Clark further said that no analyses have been made in these cases. In what sense he means "no analyses" is difficult to say. Runge's case is beautifully analyzed, not by the strict psychanalytic method, but by the hypnotic method. He made an interesting series of deductions, saying that the conduct of the patient was based on the masturbatory complex. I have presented an analysis of a patient, more or less in detail, in the June number of the *Journal of Nervous and Mental Diseases*, 1925, in which I have taken up the tremor, the flow of saliva and the greasy skin, and have attempted to show analytically what they all may mean. Hauptmann's extremely interesting analysis in the *Zeitschrift für Neurologie und Psychiatrie* gives in detail an introspective orientation of his own movements and his own feelings. This mode of description is not unlike the spoken method of Dr. Clark. The patient himself fancied all these feelings, where they came from, and gave his own interpretation.

There is one more point I wish to make. First, I feel that Dr. Clark was too specific when he spoke of the ego-neuroses as constituting the only type he observed in encephalitis. I do not think this is true. A great variety of stages are observable. The variations are kaleidoscopic; nobody can attempt to envisage them, but one thing needs to be borne in mind. I quoted Stärcke when he said he had been looking ever since he had been practicing medicine for what were called "normal" people. "Normal" is only a logical fiction. There are all kinds and stages of people. If one would use the "type" fictional conception, such may be arranged more or less according to various grades of psychosexual evolution. These differently developed persons may be involved in an encephalitic process with a resulting acute or residual chronic psychoneurosis or psychosis (Dr. Clark, if I heard him right, said they did not have psychoses), but they nearly all begin with a psychosis, and there are a lot of residual psychoses. The resultants are largely determined, however, by the stage of the psychosexual evolution of the person involved. If the person is from 8 to 12 years of age, like the fifty or sixty children described by Dr. Raynor, we do not expect a complete degree of psychosexual evolution to the social standard. They must remain in a narcissistic stage. This fact may serve to explain why that type of person will not be able to be treated by the ordinary transference methods of psychanalysis, but persons who have advanced to such a stage of psychosexual integration, the so-called "average" or "normal" people, can be handled just like any transference neurosis. I think Dr. Clark will agree with me in that respect. I think he has in mind chiefly these lower stages of integration. I do not mean "lower" in the sense of inferior; I am speaking from the standpoint of psychosexual evolution.

Dr. P. R. Lehrman: Dr. Clark's paper is important in this: he calls attention to a new method of managing narcissistic neuroses. At a recent meeting of the New York Psychoanalytic Society, where Dr. Clark presented his fantasy method and Dr. Jelliffe in socratic manner brought out the salient points of the technic, it appealed to us as a valuable modification of the psychanalytic method in the treatment of the ego or narcissistic neuroses. We may be justified in viewing the personality sequelae of encephalitis as ego-neurotic manifestations when we bear in mind the fact that the physical sequelae

of this illness are the release of paleokinetic mechanisms. Similarly in the psychic sphere, it is the release of the paleopsyche or the narcissistic factors which is in evidence in postencephalitic personality disturbances. A person may be in an advanced stage of psychosexual development and yet regress to the narcisistic stage as a result of encephalitis or other physical or psychic traumas. What Dr. Clark aims at is the mobilization of whatever libido he finds in his patients, and its conversion into object-libido. He is thus able to take the lowest form of the ego and build it up toward the goal of the ego-ideal. I have a case which will illustrate this. In the winter of 1920, I accepted for treatment a dentist who had postencephalitic physical and mental symptoms. He felt incapable to practice his profession, and when he first consulted me he was in the employ of the street cleaning department as a snow shoveler. He showed other evidence of regression. After a year's treatment he recovered sufficiently to establish an office, and at present is quite successful in his profession, though he still shows some evidence of the parkinsonian syndrome. I began with the freudian technic, but soon found that modifications were necessary. He obtained enough object libido to enable him to come back to the world of reality. We have seen other therapeutic measures help such patients. The Vanderbilt Clinic and Post-Graduate groups of encephalitic patients have been improved temporarily a few times. A few years ago a physician, having returned from Vienna, enthusiastically began treating these patients with typhoid vaccine and they improved for a time, but when he lost interest in that group they regressed. Then another physician treated the same group of patients, and the same thing happened until he lost interest. This raising of the patient's level will recur with the coming of other enthusiasts in some mode of therapy, and the mechanism of improvement or regression will be lost sight of unless we make use of our psychanalytic insight. In his paper tonight Dr. Clark is calling attention to this insight and also is giving us a method for the reconstruction of the postencephalitic personality changes.

Dr. Joshua H. Leiner: I would like to speak on this subject from the somatic standpoint. A child, aged 7, had encephalitis a year and a half before I saw him. Following the attacks he showed conduct disorders and character changes, together with respiratory disturbances. When in my office he would run around the room, destroy things, bite, scratch, stuff himself with food, and even urinate and defecate in his garments and on the floor. Four months after I saw him, he contracted pneumonia with high temperature. After convalescence he walked into my office with his gloves on his hands and a little walking stick, and deported himself like a little gentlemen—a very dramatic change. The family doctor tells me that the child a year later reverted to his conduct disturbances. This child improved because of certain somatic changes and improvement in his brain cells. I cannot correlate definite pathologic symptoms and improvement, as in this case, with a therapy such as psychanalysis. I can only speak of this case as a shock reaction occurring on the basis of foreign protein therapy. If Dr. Clark can explain this boy's improvement with the psychanalytic method, I would like to have him do so, but I cannot see the bridge that will span the gap. After any shock therapy the patients will get better, but will revert to their symptoms again after a while.

Three or four years from now, if Dr. Clark's patient will show improvement and no reversion, then we can speak of a cure, but otherwise it is only another type of therapy which probably will also be of temporary help.

DR. IRVING SANDS: Working in neuropsychiatric clinics every afternoon of the week, I am called on to treat all types of encephalitic cases, and all sorts of postencephalitic states. Many of the patients I have followed for years in the outpatient clinic of U. S. Veterans' Hospital no. 81. It is natural, therefore, that I should have formed an opinion about the subject in general. I am sorry that it is a pessimistic one.

In the largest number of instances, epidemic encephalitis is not a definitely acute condition with a decided termination. Most of the cases are of the subacute type. When there is apparent clinical recovery, ectodermic and mesodermic replacement of the destroyed tissue occurs anatomically. There is, therefore, a constant change in the anatomic condition of the brain, and consequent clinical changes. We have tried almost every known sort of therapy, but without real improvement at the end of any given time. Typhoid vaccines, mixed vaccines, iodides, and a milk albumin preparation were given trials, but none have proved what we hoped they might. We have used physiotherapy in our efforts to help these patients; but it too has proved of no real value. That does not mean that the various methods of therapy are useless, for they do help, but not to the desired extent.

It is the everyday clinic case and even office case that presents serious problems. Take the postencephalitic child who keeps the family awake at night. The wage-earner is deprived of sleep and a vicious circle is established. The creation of a center of therapy at Kings Park State Hospital is a Godsent gift for a few families. Many judges will refuse to commit children to a state hospital, and many families will likewise object. The problem is not easy. We have attached to our clinic psychanalysts of acknowledged proficiency, and yet they have had little success in handling these patients. Another problem is the impotency which is so common among the male patients; nature seemingly attempts to limit the offspring of these persons. Anybody leaving this room with the feeling that there is a definite concrete effectual means of managing these patients is greatly mistaken.

DR. GREGORY STRAGNELL: My chief interest in the subject is not the amelioration or the care of the conduct disorders of these cases or groups of cases; but I think that the following out of some such plan might help a great deal in giving us some clue by which to rearrange the conduct disorders of the bulk of the medical profession, which I think is far more important.

It seems to me that the important thing to derive from a plan which follows the lines Dr. Clark has worked out would be to follow a subject which has become very dear to me from the inspiration I have derived from the work of Dr. Jelliffe, and that is to realize some of the pathways over which the psyche manifests itself somatically, and through which the psyche is influenced by the soma. Really to try to divide them is rather presumptuous on the part of the medical profession, not only presumptuous, but extremely childish. The most important thing is for us to lose our respect for our own selves for the moment and to realize, as I have come to realize in my work more every day, that we can listen to a patient's story and prophesy not only the conduct disorder, but just about where there is going to be a tumor, or a pyelitis; and, furthermore, to find the organic weak spots, or rather the spots where the stress is going to be laid, not only in the conduct disorders; where the soma is going to break through in an effort to find expression through those pathways, whether it is encephalitis, a breast tumor, a nasal catarrh or a sinus; and those of you who insist on making gods of your microscopes and goddesses of your test tubes are going to be very much in the position that the ancients

were with the multiplicity of their graven stone images. We too little realize that the organism is a unit of a larger unit; namely, the experiences which man has passed through from the time chemicals, light, heat and power united in some way to form a living cell. Life is experience, or function, and we will not be physicians in the true sense if we think only in terms of structure.

DR. CLARK: Although we have had encephalitis as a continued topic for scientific discussion for several years, my effort here tonight is the first genuine psychanalytic attempt to explain the mental symptoms. Frankly, I did not enter this field with any great hopes of radically removing the mental sequelae of the infectious process. The main purpose was to re-enter the field of the organic neuroses by the instinctual route rather than by a neurologic or biologic one, as I did in the tics several years ago, and as I interpret so much of Dr. Jelliffe's work still to be. While both methods have their advantages, I hold that the strict psychanalytic one is the better, as Freud and Ferenczi have shown or at least suggested. I admit that I took as the paradigm of mental sequelae the worst possible types of ego neuroses in conduct symptoms; not that there are not many others of the transference type, but we see in many reports how the latter are reduced to a comparatively recovered state of mind, while no one as yet has made any encouraging permanent change in the intangible type which serves as the subject of my thesis. As I think I have made clear in my paper, we succeed in entering the unconscious conflict in our cases by giving libido to our patients instead of extracting it as in the transference neuroses. In the end we must expect that these children will objectivate their libido, but at first in order to break down the narcissistic protection to the imprisoned ego libido we have to give libido. We help them to organize their own lives about the soundest part of their still intact ego nucleus and to absorb our identified interest (libido) into the hegemony of their ego. The method of advancing this latter aim is the keynote as to whether or not the benefit gained by the patient shall remain permanent and stable. How to raise the power of ego integration is the great problem. It may not be counted as an essential defect in the nature of the ego until we have broken down all the narcissistic protection not immediately required to make the ego operative for normal living. My paper is a contribution to the psychanalytic instinctual reintegration of the ego libido rather than a structural and neurologic approach to this problem, as in the past.

SUBACUTE EPIDEMIC ENCEPHALITIS. A CASE SHOWING AN UNUSUALLY WIDESPREAD DISEASE PROCESS. DR. JOSEPH SALAN (by invitation).

I bring before you a case presenting a striking group of clinical manifestations and a rather uncommon clinical course. The patient was under almost constant observation of competent neuropsychiatrists, so that the sequence of events was studied with great care and recorded in detail. Not without interest also is the fact that the suggested diagnostic possibilities were many and varied. Among those considered were: general paralysis, cerebral arteriosclerosis with multiple areas of softening, Wilson's disease with psychosis, atypical form of multiple sclerosis, extensive chronic encephalitis and a frontal lobe neoplasm involving the right side of the brain.

J. B., Russian, aged 40, married, was a skilful mason, a good earner and of fair intellect. He had been a constant reader of books. His family history is unimportant. He was always well and was in good health directly preceding the time of onset of his present illness.

He was brought to the Neurologic Clinic of the outpatient department of Mt. Sinai Hospital on April 25, 1925, and the following history was obtained. About one and one-half years prior to his admission to the clinic he awoke one morning complaining of intense headache and dizziness. Immediately following breakfast, he vomited, became confused and failed to recognize people about him. His conduct became peculiar and childish; he would cry without provocation, or he would become mute and refuse to talk; he would move about restlessly and aimlessly, kissing various objects about the house indiscriminately. He became very clumsy in all his movements. There were, however, no convulsive attacks, no loss of consciousness, no paralytic phenomena and apparently no febrile state. He remained at home for about two days, becoming progressively worse, and was taken to the Psychopathic Division of Bellevue Hospital for observation.

The observations there are recorded as follows: Patient is confused, childish, crying at times without good reason; speech is slow and slurring; answers are frequently irrelevant and incoherent. He is disoriented for time and place; memory is impaired for remote and recent events; judgment is poor, and there is no insight. Pupils are irregular but equal; both react sluggishly to light but promptly in accommodation. There is a coarse tremor of facial muscles, tongue and fingers. There are also twitchings of muscles of the left extremities. Knee jerks are diminished; heart sounds are muffled; liver is enlarged; blood pressure is 165 systolic, and 100 diastolic; and there is "evidence of marked arteriosclerosis." The fundi are negative. The cerebrospinal fluid is negative in all phases. The blood Wassermann reaction is negative.

The patient remained at Bellevue Hospital for one month and nine days and was then committed to the Manhattan State Hospital without a definite diagnosis being offered except for the suggestion that marked arteriosclerosis was present. While at Manhattan State Hospital, he was at first very restless, displaying twitchings of various muscles, face and hands, and marked mental deterioration; he laughed a good deal without reason, and was childish in conduct and very untidy. He was confused, incoherent, disoriented and perseverated in his speech. He was helpless, and had to be assisted in dressing (very likely because of apraxic disorder). The neurologic status is recorded as follows: parkinsonian facies; pupils equal but irregular; right pupil reacts promptly to light, left is sluggish to light, both react promptly in accommodation; knee jerks are increased, left more than right; the abdominal reflexes are present; there is some resistance to passive movements; speech is dysarthric. A roentgen-ray examination showed enlargement of the liver. The biologic tests of the blood and the cerebrospinal fluid were negative in all phases. He received no antisyphilitic treatment in his ten months' stay, but showed gradual mental and no physical improvement. He was then placed in the care of his family. The diagnosis on discharge was psychosis with Wilson's disease.

Shortly after his return home he was seen by a neurologist who referred him to our clinic in the outpatient department. The findings on the first examination in the clinic were as follows: pupils irregular, unequal, right larger than left, both react sluggishly to light but well in accommodation. The eye movements could not be tested for lack of cooperation. The lower jaw is carried to the left on opening of the mouth. The face is fixed, with a washed out expression, and shows asymmetry. The tongue deviates to the left. The speech is dysarthric and monotonous. There is marked incoordination

of movements with loss of associated movements in the upper extremities; generalized hypertonia; generalized hyperreflexia, more marked on the left side. All superficial reflexes are present. Bilateral Hoffman but no other pathologic reflexes were found. There were no sensory changes.

The mental examination which, for lack of cooperation, was limited showed his memory to be intact. Spontaneous speech was partly intact. There was definite limitation in the choice of words. Speech was slurring and dysarthric. He repeated words spoken to him correctly. He understood commands spoken to him but could not carry them out correctly. He could not write spontaneously, and could not copy written letters nor write on dictation. He perseverated in movements in attempts to carry out different commands. He imitated movements with occasional error.

The blood pressure was 210 systolic, 90 diastolic; blood Wassermann reaction, 4+. At this time the diagnosis of an extensive chronic encephalitic process or right frontal lobe neoplasm was considered.

The patient remained under observation over a long period of time, and was examined repeatedly, little change being observed in the physical findings, while he continued to improve mentally. He received a short course of mixed treatment, probably not more than 1 dg. of mercury. Iodides were given for a somewhat longer period, and a course of sodium cacodylate injections was administered. A second blood Wassermann test in August, three months after the first examination, was reported negative.

His present status can be summed up briefly by saying that very slight change has occurred in the objective neurologic findings since the first examination, and that the outstanding features are: irregularity and inequality of the pupils; spastic fixed and asymmetric facies; deviation of the tongue to the left; paresis of the left upper extremity; tremors of the outstretched hands more marked on left; tremor of the head; generalized rigidity and generalized hyperreflexia, more marked on the left side; active abdominal and cremasteric reflexes; bilateral Hoffman sign; clumsy gait; somewhat slurring speech; manifestations of ideational apraxia in various fields, including alexia, agraphia and akinetic apraxia in the paretic extremity; astereognosis without sensory disturbances.

A mental examination shows him to have gained ground in many intellectual fields. His memory, orientation, judgment, insight, coherency and relevancy of speech have markedly improved. Emotionally he is also more stable.

Two important facts stand out most prominently in the clinical picture presented: (1) The abrupt onset in a healthy person of a stormy and extensive disease of the central nervous system, with the clinical manifestations of an acute dementia, characterized by psychomotor hyperactivity, emotional instability and rapid deterioration of intellect, in association with distinct organic neurologic disturbances in the nature of nuclear, extrapyramidal and pyramidal tract signs and symptoms; (2) the gradual upward progress in the condition of the patient leading to slight improvement in the physical status and fairly marked clearing of the psyche.

What are the extent and localization of the disease process, and what the character of the lesion?

The objective findings in the case unquestionably indicate a diffuse pathologic process which very likely did not spare much of any part of the brain substance. The marked intellectual disorder, the emotional disturbance, and the akinetic apraxia in the paretic arm place the disease process in the frontal

lobe, while the definite evidence of ideational transcortical apraxia with alexia and agraphia indicate a diffuse and extensive lesion in which the parietal lobe plays an important rôle. The rigidity, tremors and myoclonic twitchings (which have been present in the early phase of the disease) incriminate the basal ganglia and the midbrain area, the latter very likely being responsible also for the ocular changes. The generalized hyperreflexia with paretic phenomena indicate mild pyramidal tract involvement. Now, with such a widespread lesion what need is there and what hope for success can there be for circumscribing the disease process?

The other question, the nature of the pathologic process, is still more difficult, and can best be answered by an analysis of the diagnostic possibilities that were suggested.

General paralysis was dismissed in the face of negative serologic findings and because of the abrupt onset of a very profound dementia without prodromal symptoms and without other earmarks of general paralysis.

In favor of arteriosclerosis was, of course, the hypertension and the enlarged liver, but again the widespread character of the disease process, the lack of any tendency to focalization and the fact that the marked mental deterioration was out of proportion to the physical signs, spoke strongly against such a pathologic process.

Wilson's disease with psychosis was based mainly on the pallidostriatal signs in association with the enlarged liver. The character of the tremor, the lack of constant progression in the face of a very apparent remission, does not permit grouping the case with instances of progressive bilateral lenticular degeneration.

None of the signs typifying multiple sclerosis are present in this case.

There is a lack of evidence of intracranial pressure or any of the cardinal signs of cerebral neoplasm.

Several features in this case are common to Alzheimer's disease, such as the intellectual deterioration with disseminated and diffuse organic manifestations. But in Alzheimer's disease the onset is insidious at first, the disease process gaining momentum rapidly, and leading to a rapidly fatal issue without remissions.

For some time the patient has been regarded as having a form of extensive chronic encephalitis. It is this diagnosis that I ask you to consider along with another diagnostic possibility, the spastic pseudosclerosis of Strümpel. The latter was recently briefly described by Jacob, who in his article on extrapyramidal system disease says: "I have also described *"spastische pseudosclerosis,"* peculiar types of cases in middle age, without clear etiology, with remissions, rapid progress, and the following cardinal symptoms: beginning with anxieties, mild confusions and mild extrapyramidal symptoms, then gradually increasing in severity to a Korsakoff syndrome, with delirium anxiety, confusion and optic and acoustic hallucinations. Besides these cortical symptoms, there are pyramidal tract symptoms, such as absence of abdominal reflexes, and occasional Babinski and Oppenheim signs, but no marked paralysis. Of special note are the extrapyramidal symptoms in the form of tremor and shaking, increased tension in the extremities, akinesis, dysarthria, astasia-abasia and frequently marked pains."

There is a striking parallelism between this clinical picture and that presented by my patient. And, of course, while this case may indeed belong to this group of spastic pseudosclerosis, I would not like to be forced to discard

the diagnosis of diffuse subchronic encephalitis; for can any one tell during the life of a patient whether a given diffuse process is degenerative in character, as in pseudosclerosis, or inflammatory in nature, as in encephalitis? This can only be determined in the course of time.

DIATHERMIA IN THE TREATMENT OF MULTIPLE SCLEROSIS. DR. JUNIUS W. STEPHENSON.

Two cases of multiple sclerosis were described, which showed improvement with diathermia. The cases were of the spinal type and both showed sensory changes revealing a spinal level. The cases showed typical multiple sclerosis findings, such as diplopia, nystagmus, pallor of the optic disk, loss of abdominal reflexes with spasticity. One case was that of a woman, aged 25, whose symptoms had existed for about two years. This case showed a Brown-Sequard syndrome with the pain and temperature level on the left side at the sixth thoracic vertebra gradually shading up to the seventh cervical where it was within normal limits. The sense of position in the right toes was completely lost.

The second case was that of a man, aged 50, an advanced case of at least eighteen years' duration. He showed a bilateral sensory level at the eighth cervical with loss of position sense in all toes. Vibratory sense in this case was much reduced.

In the first case manometric test showed a distinct block, whereas in the second there was no evidence of block. A pad 8 inches (20 cm.) long and 2½ inches (6 cm.) wide was devised and in the first case was applied from the seventh cervical down and in the second from the fifth cervical down. In both cases 800 milliamperes for a period of twenty minutes was the first application. Whereas before the treatment neither patient could recognize the position of the toes, twenty-four hours after treatment the first patient made correct answer in 50 per cent of the test, and three days later a perfect response. At the same time there was a distinct diminution of the pain and temperature impairment which in the course of ten days had entirely disappeared. In the second case, twenty-four hours after the treatment, the patient made only one mistake in the position of his toes, and forty-eight hours later, none. In this case the sensory level, in the same period of time, dropped from the eighth cervical to the ninth thoracic segment. The first patient has been kept under close observation. There has been a progressive improvement in her power of locomotion, until, six weeks after the first treatment, she went for treatment alone, in the couse of her journey having to climb elevated and subway stairs. Before treatment she could not walk alone. Also, the first bedside note as to her improvement was made three days after the first treatment. For four months the treatment was given three times a week. There was a gradual increase in the dosage until she was receiving 1,400 milliamperes for twenty minutes. Five months after treatment she could walk a straight line and did not sway with the eyes closed; finger to nose and knee to heel tests were perfect. All the abdominal reflexes had returned except the right lower but were exhaustible. She still showed a suspicious Babinski sign, though there was no suggestion of the previous inexhaustible ankle clonus. I feel that as in the two cases there was identical behavior in the sensory findings occurring within the same interval of time, this was not a coincidence nor remission, but an actual result of the therapy which justifies further investigation. Case 1, showing a distinct block, proved there must have been some swelling of the cord, and this was most probably of an inflammatory and not

degenerative nature, and that the effect of the heat of the diathermia produced an increased vascular supply, thereby increasing the protective forces at this particular lesion. The prompt response observed in the sensory fields was probably due to the fact that such sensory changes are not so common, that these tracts are less vulnerable and therefore respond more promptly. Oppenheim said that in the majority of cases of multiple sclerosis, if the cases are sufficiently closely observed, sensory findings, though fleeting, can be demonstrated, and we should put more effort to find such changes and apply treatment accordingly.

DISCUSSION

Dr. Joseph H. Globus: I think Dr. Salan in his report covered the ground, and so well that little room is left for discussion. Apparently the patient continues to improve, for there is little of the head tremor left, and the tongue deviates but slightly. He demonstrates almost every form of apraxia: motor, akinetic, ideational, etc., which must be regarded as evidence in favor of a diffuse lesion, most likely inflammatory in character, with secondary degenerative changes. Of the latter, of course, one cannot be certain.

Dr. Jelliffe: I have one word to say, now that Dr. Stragnell has given me an opportunity, about a behavior disorder quite frequent in doctors. It is a pernicious disorder. If I may speak from my observation at medical meetings, it consists in a devotion to "names" of things, rather than the behavior of "things." We have heard about "multiple sclerosis" and the treatment of "multiple sclerosis" by diathermia. Of course bacteriology came in as a blessing, but it also came in as a curse, because from bacteriologic deductions our conceptions of diseases took on very definite aspects. Thus there was "scarlet fever," "measles," "smallpox" and "syphilis" — clear-cut conceptions of which may be said to constitute "diseases," but, on the other hand, the extension of this conception beyond its legitimate rôle constitutes the behavior disorder of the doctors of which I speak. There is no such thing as "multiple sclerosis" in the same sense that we speak of a thing like measles. It is not a disease process *per se*. Multiple sclerosis pictures are produced in encephalitis; from tumors; from blood diseases; from syphilis, and from a number of other things. At least fifteen or twenty conditions are to be found in the literature which show the multiple sclerosis picture. There are fifteen or twenty types of process which result in disseminated blocking of certain neurons. What has diathermia done for the particular form under consideration — not for the fiction "multiple sclerosis"? That is a legitimate inquiry, and I think Dr. Stephenson has brought up an important point. Lhermitte preceded him in the treatment of these so-called multiple sclerosis syndromes, as well as in syringomyelic processes by roentgen-ray and diathermic methods. It is important in these syndromes, for which practically nothing is being done, to get some idea of the relationship between the action of what diathermia may be supposed to do and the type of lesion which may be supposed to produce such blockings in the functional pathways of the spinal cord. In that sense Dr. Stephenson has brought us something worth thinking about, and he has also suggested the possibility of the type of pathologic lesion in one of the cases. I feel a little suspicious regarding the interpretation of the action of diathermia in a chronic progressive process which has been going on for a number of years, when improvement begins within twenty-four hours.

As to the other case, that of Dr. Salan, I have not much to offer, except along the same line. An encephalitic process can result in a multiple variety

of mosaic-like pictures, like the bursting of a shrapnel shell, as Marie once said of the action of the encephalitic virus. It spreads all through the brain, the basal ganglia and the medulla, and gives rise to a multiple series of syndromes. Were there any tests of liver function done, the hemoclastic crisis, for example?

DR. SALAN: No.

DR. JELLIFFE: The liver picture and the arteriosclerotic picture are of great interest. I have spoken frequently of the relationship between the vegetative syndromes, the involvement of certain synapse junctions in the upper reflex arcs as they are integrated in vegetative functions. That is so in Wilson's disease. When I read his paper I wrote to him and said: "Here is a great opportunity to learn something about the vegetative nervous control of liver function." He never could see the point. He wanted to assume that the liver toxin produces a localized lesion in the basal ganglia, which may be so, just as carbon monoxide poisoning has a localizing predilection for related structures. I wanted him to look at the problem from the standpoint of the interruption of the integration of the body as a whole, whereby pathways coming through the basal ganglia in the region of the third ventricle and in the mesencephalic region were interrupted, and that such interruptions can produce diseases of the viscera, such as arteriosclerosis and changes in the liver, diabetes, etc. This latter view has now been abundantly proved.

DR. GLOBUS: In apraxia, the patient who displays such disturbance, must necessarily understand the act he is to carry out. Whether he is an idiot or whether he is a genius it does not matter. An idiot may light a cigaret well, while a genius may fail to do so, if the latter has some form of apraxia. The important thing is that the person examined must comprehend the act he is asked to carry through. This patient does understand it; he is not an idiot, nor, of course, a genius. He is capable of carrying out only some very simple acts.

I cannot fully agree with something Dr. Jelliffe has said. I must confess that I am an organic neurologist and cannot see how the other extreme of neurology can undertake to solve with their methods some of the problems which are put to the organic neurologist, as for example, the localization of cerebral tumors, in which he is to depend mainly on localizing signs, and must accept as a working hypothesis the belief that certain parts of the brain carry out certain functions independent of other portions of the brain. Particularly we must be allowed to speak of neurons, if the other group of neurologists continue to speak of synapses so loosely.

DR. JELLIFFE: You assume that there is such a "thing" as a center.

DR. GLOBUS: Yes, there is such a thing as a center, if we regard the latter as a circumscribed group of nerve cells subserving a common function.

DR. CLARK: I had supposed, as the school of psychology had given up a special and distinct faculty formation of the mind, that neurologists had long since discarded the strict brain localization also, and that one possessed no more logic or good physiology than the other. Many will remember Franz's review last year of the strict localization hypothesis of brain function, and his indication that it was no longer tenable; while in the main it was still to be used for diagnostic purposes, the brain acting as a whole in all of its special functions was really the important principle to be borne in mind. Dr. Globus' statement, therefore, that there is a hard and fast rule in regard to the neuron conception being a surer comprehension of physio-anatomic methods of under-

standing brain function, has really no better substantiation than a synaptic theory or the psychanalytic conception of the libido formation of the whole nervous system. They are but different ways of thinking and understanding the anatomic and functional aspects of the nervous system in the living organism of the individual. We must be careful, however, not to mix our categories, as both have their validity, and the question is, which is the more useful in our understanding special disease process?

DR. GLOBUS: I must take exception to the statement of Dr. Clark and insist on the usefulness of the neuron idea. We are constantly meeting problems demanding the determination of whether an upper or lower neuron is involved in the disease process. In such instances the conception of the neuron theory is absolutely indispensable. On the latter we must often fall back, as, for instance, when we wish to determine the seat of a lesion causing various forms of paralysis, flaccid or spastic.

DR. JELLIFFE: Nobody denies that; there is no controversy there. Dr. Globus says that in peripheral paralysis a spinal cord center is involved. In hysterical peripheral paralysis where is the cord? Nobody denies the value of the conception of what are called centers. I think the conception of what are called "foci of synapses" is better than "centers." I think Dr. Clark has pointed out the availability of a possible hypothesis that will explain the situation. Sticking too closely to the conception of neuronic centers we are lost the minute we compare an hysterical paralysis with an anterior poliomyelitis paralysis, except so far as in the one the reflexes are not gone, and in the other they are gone. Dr. Globus says he is a pure neurologist. If he likes centers, I hope he will buy von Economo's latest atlas so he can see how complicated they are.

DR. STEPHENSON: I have nothing to add except in reply to Dr. Jelliffe's apparent criticism of my veracity. The marked improvement within the twenty-four hours was in the sensory findings and not the motor, and as I stated, I thought that might be explained on the theory that sensory findings to the degree here shown are not common, which means that these tracts are less violently attacked and in consequence respond more promptly to therapy. I am sure in his large experience Dr. Jelliffe has seen just such changes, not only in multiple sclerosis but also in syringomyelia, follow an ordinary lumbar puncture.

NATHAN E. BRILL, M.D.

WHEREAS, in the loss we have sustained by the decease of our esteemed colleague, Dr. Nathan E. Brill, we, the members of the New York Neurological Society, are desirous of testifying our respect for our departed fellow-member, and of expressing our heart-felt sympathy with the family in their great bereavement, and with the medical profession on the loss of one of their ablest and most enlightened fellows. Therefore, be it

Resolved that we express our sincere condolence to the family of our deceased confrere in this, their hour of affliction.

Resolved that the New York Neurological Society has lost one of its most valued and respected members.

Resolved that the entire medical profession of which our departed fellow-member was a shining light has sustained an irreparable loss.

Resolved that this expression of our sentiments be dully inscribed on the records of the New York Neurological Society, and that a copy thereof be transmitted to the family of the deceased.

Book Reviews

THE FAITH, THE FALSITY AND THE FAILURE OF CHRISTIAN SCIENCE. By WOODBRIDGE RILEY, PH.D., Member of the American Psychological Association. Lecturer at the Sorbonne, 1920. Author of "American Thought from Puritanism to Pragmatism." FREDERICK W. PEABODY, LL.B., Member of the Massachusetts Bar. Author of "The Religio-Medical Masquerade." CHARLES E. HUMISTON, M.D., Sc.D., Professor of Surgery, College of Medicine, University of Illinois. Price, $3.50. New York: Fleming H. Revell Company, 1925.

Each of the authors of this volume has treated a single phase of the complicated picture presented by Christian Science, the opening chapters being devoted to a careful analysis of the various sources from which this system of "so-called" religious healing has drawn its inspiration. Dr. Riley, from investigations which have covered every printed article on Christian Science, its own publications and all of those articles which have had a bearing on this subject, shows that the origin, inspiration and even the name were derived from the vaporings of "the Portland Mesmerist, Quimby." He clearly proves this by documentary evidence showing the gradual evolution of "Mother" Eddy, her first halting steps toward independence, the gradual assumption of self-consciousness and finally the inevitable attempts to belittle, patronize and suppress the real source of her intellectual vagaries, Quimby's writings being referred to as an "unformed mass of illiterate speculations."

The early chapters describe the discovery by Quimby of his system, his mesmeric principles, his hypnotic trances and the induced exaggeration of sight and hearing, true hysterical phenomena, his assumption of the rôle of priest, magician and physician, the primitive medicine man, and his own nominating of his system "Christian Science" in 1863. This system was mental medicine, a manual of suggestive therapeutics rendered magical by the added principles of telepathy and thought transference, flavored with occultism and clairvoyance. The greatest absurdity—the production of disease by its being named—is transplanted in toto from Quimby's writings.

The energizing influence which entered Christian Science following the death of Quimby in 1866 is shown to have come from the "visionary vegetarian," Bronson Alcott. From this metaphysician was gleaned the immaterialism which so thoroughly permeates the gospel. Such examples as "the illusion of matter," "the body as the error of mortal mind" and "the soul as a lapsed emanation of the deity," demonstrate the extent to which Mother Eddy "borrowed" in the development of her "original" doctrines.

The third source of inspiration was Mother Ann Lee, the Shaker seeress. The guiding hand of this sect is "The Holy, Sacred and Divine Roll and Book of the United Society of Believers," and a perusal of it discloses the source of many of the principal ideas embodied in Christian Science—the mother complex, the motherhood of God and the "mother in Deity." The facts of these plagiarisms, particularly from Quimby and Alcott, are presented in considerable detail, and so conclusively that no doubt can be entertained as to the complete unoriginality of the entire structure of Mother Eddy's Christian Science. "Eddyism is Alcottism reflected in a muddy mind."

The paranoid trend in Mrs. Eddy's psychologic processes may be seen in the development of her malicious animal magnetism, a form of demonology which became a haunting dread, dogging her steps, causing her later to blame the petty annoyances of urban housekeeping on malicious animal magnetism. This is clearly seen in her claim that her husband had died of "mesmeric poison," whereas the necropsy showed that he died as the result of chronic, cardiac valvular disease.

Periods of amnesia, automatic writing and trance states in her early life have been described, while in her later years she suffered from visual and sensory hallucinations, delusions and obsessions. These psychotic phenomena stamp her indelibly as at least a psychopathic personality.

There can be no question of the simultaneous or independent development of broad, underlying generic principles which might have honestly given origin to two unrelated, although philosophically similar, systems; for the contacts, the priorities and the fatal parallels are presented and conclusively explode any possible argument in favor of the authenticity of Christian Science as originated by Mother Eddy.

The second section is written by Mr. Frederick W. Peabody as a supplement to his already published exposé of Christian Science, "The Religio-Medical Masquerade ." He shows the autocracy built up by Mrs. Eddy, "which tolerates no shadow of rivalry—demanded and received implicit, unquestioning, instant obedience." The entire structure of the church and its governing constitution and by-laws demonstrate one of the most perfect systems of exploitation and the most depressing examples of servile subordination which grace the pages of history.

At the death of Mrs. Eddy, the board of directors became the governing body of the Church of Christ Scientist, through which event a remarkable transformation took place—this body demonstrating its capability of being as autocratic after her death as it was servile before that "catastrophy." Mr. Peabody briefly sketches the personnel of this board, their weaknesses and foibles, and the official findings of a court of law arising from a suit between two members of the board.

An ominous but at the same time illuminating chapter concerns itself with activities in the suppression of education which might tend to discredit the system or expose its source, method or control. Mr. Peabody's writings have disappeared from the bookstores and libraries, the pamphlets being stolen and destroyed, and the booksellers intimidated. McClure's Magazine carried out a most complete investigation of Mrs. Eddy's life, appearing first in serial form and later as a book. This history has disappeared from the bookstores and the publishers refuse to fill orders.

A careful, critical study of Christian Science by Dr. Riley was published in "The Cambridge History of American Literature." It duly appeared and 1,500 copies were sold. Something happened—the edition was suppressed and the copies that were sold were, wherever possible, recalled.

In the chapter entitled "Lies," Mr. Peabody categorically quotes twenty statements together with the facts which demonstrate their falsity. In the chapter entitled "Death," the ever-recurring refutation of all that Christian Science teaches is shown stretching out its hand for the directors and finally the founder of this cult—the "Grim Reaper" displaying but little respect for this last final absurdity of Christian Science.

The worldly wisdom of Mrs. Eddy is clearly shown by the system of imposts and levies both direct and indirect which she built up for her financial support. Her pecuniary control was absolute; the by-laws commanded the purchase of books, manuals and periodicals; and through these forced and free-will contributions, an annual net profit of more than $500,000 rolled in. A detailed description of some real estate deals intimates that Mrs. Eddy was the gainer by $6,000 and her parishioners the voiceless losers by the same sum.

In his concluding chapter, "Cash," Mr. Peabody demonstrates that when Mrs. Eddy founded Christian Science, herself over 60 years of age, she was practically in reduced circumstances, while at her death she was possessed of $3,000,000.

Encouraged by the success of "Science and Health," Mrs. Eddy produced "Miscellaneous Writings" and enjoined all her teachers and readers to stop teaching and sell books for a year. A touch of the ludicrous is added to the picture of grasping avarice by the statement that she requested the faithful to send her three new jackets, stating that "All may contribute to these." How many dollars were realized by this little by-play is unknown! The sale of the plated mottoed spoons shows that no avenue for revenue was too small or petty to be exploited.

The third section is written by a practicing surgeon. Many psychologic and psychiatric analyses of Christian Science have appeared from time to time during the past quarter-century and Dr. Humiston does not through his contribution add materially to our comprehension of this "therapeutic system"; but he serves well by opening its records to medical analysis, crucifying its claims and pitilessly exposing its failures. The continued refusal of its members to submit to the same legal requirements as those imposed on the practitioners of other systems stamps this therapeutic system as a sham. Its sotto voce admission that surgical conditions may at times be better treated by surgeons shatters its whole structure of mental error.

Dr. Humiston points out that Christian Science through its failure to recognize acute surgical conditions, communicable and infectious diseases, its resistance to sanitary precautions and to the efforts to restrict the spread of infectious diseases, its heart-breaking, callous indifference to the suffering and death of helpless, innocent children and incompetent adults, constitutes a menace which strikes at the health and sanity of the entire body politic. Dr. Humiston rightly indicates that the cures of Christian Science are among conditions which have a death rate of zero—for it is the psychoneurotic and the psychasthenic patients who lift up their voices in adulation of the "Mother Church" and its therapeutic triumphs. The palpable absurdity of a system of healing elaborated by a faith which denies the presence of disease and the constant contradictory statements which fairly bristle through "Science and Health" brand the entire structure as a tissue of fallacy. For example: "the blood, heart, lungs, brain, etc., have nothing to do with life." "Christian Science heals organic disease as surely as it heals that which is called functional; a physical diagnosis of disease—since mortal mind must be the cause of disease—tends to induce disease." "Realize that the evidence of the senses is not to be accepted in the case of sickness," and so on. No better example of the futile and puerile processes of reasoning can be quoted than the following: "The daily ablutions of an infant are no more natural nor necessary than would be the process of taking a fish out of water every day and covering it with dirt in order to make it thrive more vigorously in its own element."

Chapter III contains a series of abstracts purporting to represent the types of disease amenable to Christian Science therapy. A perusal of this series soon indicates that glittering generalities, buncomb and lying misrepresentations characterize all of these testimonials and make careful refutation unnecessary and impossible. Such evidence would be laughed out of any medical court or tribunal.

Chapter IV contains a careful and restrained statement of the proved failures of Christian Science which were contributed to Dr. Humiston as the result of a careful questionnaire sent to the leading members of the medical profession throughout the country. The convincing contrast between the gibberish of the science testimonials and the careful, measured and substantiated medical attestations of incompetency, failure and ignorance will convince all except the blindly bigoted and prejudiced supporters of the cult. The proofs offered by Christian Science are so flimsy, so unconvincing and at times so palpably dishonest that one is at a loss to recognize the necessity for refutation. The utter ridiculousness of the entire system seems so patent that disproof would seem to be a task of supererogation.

This arraignment raises an issue which demands a conclusion and can terminate only with the complete refutation of the charges contained in this volume or the dissolution of Christian Science as a medical therapeutic system. It has already been declared a business and not a religion. This book should find its way to the desk of every legislator in every federal, state, county and municipal body.

Christian Science, progress and evolution are incompatibles. It is a commercialized system of exploitation, conceived in dishonesty, developed by grinding selfishness, displaying on the one hand servile abnegation and on the other arrogant autocracy; a so-called religious movement, its sources repudiated, extracting from its members millions of dollars, dispensing its worthless healings at fabulous prices and spreading its poisonous breath over helpless children and deluded adults. It not only takes its place as one of the philosophic comedies of history but also as one of the most atrocious tragedies in this play called Life.

LE ROMAN D'UNE ÉPIDÉMIE PARISIENNE. LA KLEPTOMANIE? By A. ANTHEAUME. Pp. 225. Librairie Octave Doin, 1925.

This book deals with the subject of kleptomania, a word, according to the author, which serves as a motive and excuse to steal, and which, passed from mouth to mouth, has created among the women of Paris during the last thirty years an epidemic of thefts in the large shopping centers of the French capital. After more than twenty years in the medicolegal field M. Antheaume is convinced that no such thing as kleptomania exists and that it should be eliminated from the medical list of nervous afflictions, an opinion substantiated by numerous inquiries and investigations at the large Parisian shops, and confirmed at medical meetings in Paris.

The author points out the difference between thefts committed by alleged kleptomaniacs of Paris and thefts committed by persons really irresponsible, such as some who are epileptic, defective, etc. He claims that the epidemic is the result of the mental attitude of the French public and that a similar condition is unknown in any other large city in the world. He details the various methods used to guard against such thefts in other countries, and favors the system used in Buenos Ayres, where inspectors are placed at all display

counters who narrowly watch all customers so that persons tempted to steal immediately feel themselves under observation and naturally refrain from doing so. He advises that a similar system be adopted in Paris; that better class women yielding to the temptation of appropriating desirable things displayed before them be more severely dealt with than their poorer sisters who lack the money to purchase; that women caught in a first offense be leniently dealt with and dismissed with a caution; but that on a repetition of the offense they be brought before the courts with full publicity, and be held entirely responsible for their act; they should be judged from the penal and not from the medical standpoint; only in this way can this blot on the social life of Paris be stamped out.

HEREDITY AND ENVIRONMENT IN THE DEVELOPMENT OF MEN. EDWIN GRANT CONKLIN, Professor of Biology, Princeton University. Fifth Edition Revised. Pp. 344. Princeton University Press, 1923.

This is the sixth edition of this excellent book, undoubtedly, the best short work on the subject. It is presented in comprehensive and readable form, the parts dealing with the problem of evolution being particularly interesting in view of the recent publicity that this subject has gained in some of our courts and newspapers.

News and Comment

FELLOWSHIPS IN NEUROPSYCHIATRY

The Graduate School of Medicine, University of Pennsylvania, in cooperation with the Commonwealth Fund of New York, offers four fellowships in neuropsychiatry with special reference to child guidance. These fellowships, which are at a stipend of from $2,000 to $2,600 per annum, begin Oct. 11, 1926, and continue for a period of three years. Applications should be addressed immediately to Dr. George H. Meeker, Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia.